

44

I

W. G. D.

The Journal of Nervous and Mental Disease

The Official Organ of

The American Neurological Association
The New York Neurological Society and
The Philadelphia Neurological Society

EDITORS

Dr. CHARLES L. DANA

Dr. HUGH T. PATRICK

Dr. F. X. DERCUM

Dr. JAS. J. PUTNAM

Dr. CHAS. K. MILLS

Dr. B. SACHS

Dr. M. ALLEN STARR

ASSOCIATE EDITOR

Dr. WILLIAM G. SPILLER

MANAGING EDITOR

Dr. CHARLES HENRY BROWN

25 W. 45th Street, New York

45138
415199

VOLUME XXV., 1898

NEW YORK

No. 25 WEST 45TH STREET

1898

ORIGINAL CONTRIBUTIONS.

	PAGE
A Contribution to the Study of Secondary Degeneration Following Cerebral Lesions, by W. G. Spiller.....	1
Some Notes on Echolalia, with the Report of an Extraordinary Case, by M. W. Barr.....	20
On Arrested Development and Little's Disease, by W. G. Spiller..	81
On the Advisability of Operation in Microcephaly, by W. W. Keen..	105
Glioma of the Right Frontal Lobe of the Brain, by W. C. Krauss..	109
The Psychological Mechanism of Delusions, by W. Hirsch.....	159
Equilibration and Its Relations to Vertigo, by F. K. Hallock.....	175
A Report of a Case of Unusual Edema in Hemiplegia, by H. A. Hare	189
Friedreich's Ataxia, by M. A. Starr.....	194
Sexual Perversion or Vice? A Pathological and Therapeutic Inquiry, by Morton Prince.....	237
Dissociation of Sensation of the Syringomyelic Type: Occurring in Pott's Disease, by D. L. Edsall.....	257
A Case of Landry's Paralysis, by W. L. Worcester.....	299
Glia and Gliomatosis, by S. Flexner.....	306
Double Ophthalmoplegia Chronica Externa, by F. Peterson.....	310
On Multiple Sclerosis, with Especial Reference to Its Clinical Symptoms, Its Etiology and Pathology, by B. Sachs. 314, 464	
On Landry's Paralysis, with the Report of a Case, by C. K. Mills and W. G. Spiller.....	365
Cases of Trigeminal Spasm: Resection—Probable Presence of Sensory Fibres in the Seventh Nerve, by J. K. Mitchell.....	392
Hypertrophic Nodular Gliosis, by J. Sailer.....	402
President's Address, New Paths in Psychiatry, by F. Peterson....	444
A Case of Serous (Alcoholic) Meningitis Simulating Brain Tumor, by Theodore Diller.....	441
A Case of Universal Muscular Atrophy, by H. A. Hare.....	450
Cases of Ophthalmoplegia, by B. Sachs.....	452
A Case of Myxoedema Treated with Thyroid Extract and with Thyrocolloid, by R. H. Cunningham.....	458
President's Address, American Neurological Association, by G. M. Hammond	505
On Myotonia, by G. W. Jacoby.....	508
A Case of Amaurotic Family Idiocy, with Autopsy, by F. Peterson	529
The Pathological Anatomy of a "Fatal Disease of Infancy, with Symmetrical Changes in the Region of the Yellow Spot" (Warren Tay): "Amaurotic Family Idiocy" (Sachs); "Infantile Cerebral Degeneration" (Kingdon and Russell), by W. Hirsch	538
Pathological Report on the Eyes of Dr. Hirsch's Patient, with Amaurotic Family Idiocy, by W. A. Holden.....	550
Congenital Facial Paralysis, by H. M. Thomas.....	573
A Case of Multiple Syphilitic Neuritis, F. R. Fry.....	594
Long Remissions in Epilepsy and Their Bearing on Prognosis, by Wharton Sinkler	601
Report of a Case of Purulent Internal Pachymeningitis, Complicating Middle Ear Disease, by W. M. Leszynsky.....	609
Family Periodic Paralysis, by E. W. Taylor.....	637, 719

RC
321
J78
V.25

	PAGE.
Experimental Researches on the Localization of the Sympathetic Nerve in the Spinal Cord and Brain, and Contributions to Its Physiology, B. Onuf and J. Collins.....	661
A Summary of the Symptoms in Sixty-one Cases of Locomotor Ataxia, with Additional Remarks, by W. H. Riley.....	679
On Regeneration of Nerve Fibres in the Central Nervous System, by W. L. Worcester.....	698
On Scleroderma and Chronic Rheumatoid Arthritis, by F. X. Dercum	703
A Case of Katatonic Melancholia, by J. E. Courtney.....	747
On the Etiology and Pathogenesis of the Post-Traumatic Psychoses and Neuroses, by J. J. Putnam.....	769
On Resection of the Gasserian Ganglion, by W. W. Keen; with a Pathological Report on Seven Ganglia Removed by Prof. Keen, by W. G. Spiller.....	800
A Case of Cerebral Ataxia, Affecting Chiefly the Right Upper Extremity, with Marked Involvement of the Stereognostic Sense, by I. Olmstead.....	807
A Case of Syringomyelia and Two Cases of Tabes with Trunk Anæsthesia, by H. T. Patrick.....	837
A Case of Huntington's Chorea with Remarks Upon the Propriety of Naming the Disease "Dementia Choreica," by F. K. Hallock.....	851
The Bruce Microtome, by C. E. Riggs.....	865
A Clinical Consideration of Herpes Zoster, by L. Weber.....	868
Chlorosis and Retinopapillitis, by H. M. Bannister.....	874
Brain Tumor Simulated by Anæmia, by H. T. Patrick.....	882

INDEX OF AUTHORS' NAMES.

Figures in parentheses () indicate discussions. Figures with asterisk * indicate original articles and are accompanied with title. Figures unaccentuated, accompanied with title, indicate abstracts ; without title, book reviews.

	PAGE.		PAGE
Achard—Paraplegia	71	Biro, M.—Sciatic Neuritis.....	291
Achard, M., and Levi L.—		Bitot, E., and Sabrazes, J.—	
Transitory Paralyzes, 356,		Tabes and Sensibility of the	
Tabes and Reflexes.....	498	Testicle	499
Adams, S. S.—Choreic Em-		Bohn.—Insomnia	75
bolism	354	Bolton, F. E.—Illusions.....	357
Alexander, H.—Mental Phases		Bolton, J. S.—Technic.....	214, 347
of Tuberculosis.....	632	Bolton, J.....	(821)
Allerhand, J.—Technic.....	139	Bond, C. H.—Diabetes and In-	
Allyn, H. B.—Multiple Neu-		sanity.....	146
ritis	287	Bonnier, P.—Sense of Position.....	493
Alzheimer, A.—Colloid Degen-		Bonnus, G.—Friedreich's Dis-	
eration Brain.....	565	ease	70
Angell, E. B..... (32), (613)		Booth, J. A.—(128), (336),	
Apathy, S.....	(45)	(483), Thyroidectomy in	
Apert, M.—Bacteriology of		Graves' Disease	766, 872
Chorea	564	Borda, J. F.—Prognosis in In-	
Ayres, S.....	(34)	sanity.....	633
Babinski, J.—Reflexes.....	220	Bordier and Frenkel—Facial	
Bailey, P.—(80), (136), 635, (821)		Paralysis	289
Baker, Smith.....	(38)	Botezat, E.—Nerve Endings of	
Ball, M. V.—Paralytic Chorea.....	356	Tactile Hairs	281
Bannister, H. M.—Chlorosis		Bourdon, P.—Muscular Sensi-	
and Retinopapillitis.....	*874	bility of the Eyes.....	493
Barker, L. F.—52, 343, Epidemic		Bramwell, B.—Pseudo-Hyper-	
Cerebro - Spinal Meningitis,		trophy, 220; Aphasia.....	763
565, 826		Prower, D.—Friedreich's Dis-	
Barr, M. W.—Echolalia.....	*20	ease	72
Barrows, F. W.—Inanition and		Bruce, A.—Endogenous Fibres,	
Nerve Cells.....	283	215	
Barteit.—Thyroidine	701	Bull, S. — Lumbar Puncture.....	151
Bastian, H. C.—Aphasia.....	897	Bunzl-Federn, E. — Accessory	
Bastianelli, G.—Combined Scle-		Nerve Nucleus.....	346
roses	142	Burr, C. W.—(37), (125), (278),	
Batten, E.—Muscle Spindle.....	282	(279)	
Bechterew, A.—Letter from		Cabitto, C.—Epilepsy Treat-	
232, Eye Muscle Nuclei 346,		ment by Hot Air, 489; Tox-	
Early Symptoms of Tabes,		icity of Sweat.....	493
499, Ankylosis Spinal Cord.....	762	Cagigal & Lepierre.—Sleeping	
Bell, J.—Bullet shown by Skia-		Sickness	593
graph	566	Capitan.—Thyroid Chlorosis ..	899
Bennett.—Ulnar Nerve Injuries.....	293	Cassirer, R.—Muscle Pathology,	
Berger, H.—Anterior Horn		890	
Cells in General Paresis.....	493	Cattell, H. W.....	(42)
Berkeley H. J.—Technic.....	623	Chantemesse, M. and Marinesco.	
Biernacki.—Endarteritic Myelo-		—Tetanus	626
pathy	892		

	PAGE.		PAGE.
Charon, R., and Briche, E.—		Egger, F.—Muscular Atrophy.	223
Blood in Epilepsy.....	701	Ehlers.—Paresis and Syphilis.	147
Cheron, P.—Chorea.....	74	Elder, G.—Intracranial Circulation.....	623
Chipault.—Lumbar Puncture,		Erb, W.—Intermittent Lameness.....	891
150; Perforating Ulcer.....	293	Eshner, A. A.—(44), (208);	
Clarke.—Anemia and Spinal		Tremor.....	562
Cord Lesions.....	141	Eurich, F. W.—Neuroglia.....	490
Clemesha, J. C.—Thomsen's		Facklam, F. C.—Huntington's	
Disease.....	608	Chorea.....	352
Collins, J.—(57), (267), (337);		Faure, M.—Re-education Method in Tabes.....	501
Glioma of Medulla, 569, (607);		Féré—Hemiplegia, Infantile....	628
Sympathetic, *661, (677), (699),		Féré, Ch.—Reflexes in Epileptics.....	220
(815), (816), (863)		Finlay, F. G.—Tic Convulsif....	356
Coston.—Delirium.....	72	Fisher, E. D.—(78), (606), (815).	
Courtney, J. E.—Katatonic		Flatau, E.....	67
Melancholia.....	*747	Fleischmann.—Lumbar Puncture.....	148
Courtney, J. W.—Pseudo Tabes		Flemming, W.—Spinal Ganglia.	401
Crawford, J. F.—Temperature		Flexner, S.—Gliomatosis.*306,	
Sense.....	492	(340)	
Cunningham, R. H.—Myxœdema.....	*458	Fournier, L.—Death in Hysteria.....	19
Curtis, B. F.—(479), (480), (483)		Fraenkel, J.—(129), (266), (269),	
Daland, J.....	(752)	(336), (823)	
Dana, C. L.—(203), (336), (483).		Fressonn, H.—Paraplegia.....	71
(598), (599), (744), (805), (870)		Friedländer, A.—Facial Paralysis in Typhoid.....	889
Darkschewitsch, L. O.—Paralysis and Pasteur Treatment....	804	Frey.—M. V. Technic.....	281
Dearborn, G. V.—Imaginations.	357	Fry, F. R.—Multiple Neuritis,	
Dearborn, G. V., and Spindler,		*594, (600)	
F. N.—Motor Reactions.....	765	Gilles de la Tourette.—Treatment of Tabes by Stretching..	500
Dejerine, J.—Hysterical Contracture.....	355	Goldflam, S.—Family Paralysis.	825
Dejerine and Sérieux.—Aphasia.	824	Goldscheider, S., and Brasch,	
Dejerine and Thomas.—Alcoholic Paralysis.....	285	F.—Nerve Cells in Fever....	351
Delabarre.—Reaction Movements.....	145	Goldscheider, S., and Flatau, E.	
Dercum, F. X.—(124), (125),		—Nerve Cells in Fever.....	351
(127), 225, (273), (276). Movement of Neron, 348, (485),		Goldscheider, S., and Moxter.—Nerve Cells in Fever.....	351
(488), (558), (614), (615), (617).		Goldthwait.—Paralytic Deformities.....	633
Scleroderma, *703, (750).		Grünbaum, A. S.—Muscle Spindle.....	219
Desplats, M.—Saturnine Encephalopathy.....	77	Gudden, H.—Technic.....	139
Dignat, P.—Electricity in Hemiplegia.....	634	Habel, A.—Tabes and Floating Kidney.....	498
Diller, T.—Alcoholic Meningitis.....	*441, (527), (614)	Hall, G. S., & Allen A.—Tickling and Laughing.....	292
Diller, T., & Meyer, A.—Landry's Paralysis.....	285	Hallock, F. K.—Vertigo, *175;	
Donath, J.—Facial Hemiatrophy.....	224	Huntington's Chorea.....	*851
Drury, M., and Folsom C.—Study Effects.....	614	Hammond, G. H.—President's Address.....	*505, (527), (608)
Dukes.—Backward Children and Thyroid.....	881	Hand, A.....	(279)
Edsall, D. L.—Syringomyelic Dissociation.....	*257		

	PAGE.		PAGE.
Hare, H. A.—Edema in Hemiplegia, *189; Muscular Atrophy	*450	Keen, W. W., and Spiller, W. G.—Gasserian Ganglion.....	*800
Heinersdorff, H.—Amaurosis and Occipital Lobe Abscess.....	567	Kempf.—Uræmia	570
Held, H.—Nerve Cell Structure, 214; Acoustic Paths	281	Kiernan, J. G.—Encephalitis.....	401
Henschen, S. E.—Roentgen Rays	627	Klippel, M.—Taste and Smell in Tabes.....	499
Herter, C. A.—(54), (57), 153, (201), (815), (820), (821)		Knapp, P. C.....	(849)
Hibbard, C. M.—Melancholia and Urea.....	631	Koller, C.....	(205), (557)
Higier, H.—Optic Diseases.....	289	Krainsky, N.—Epilepsy Pathology	758
Hill, A.—Granules, 491; Thorns.....	561	Krauss, W. C.—Glioma Frontal Lobe	*109
Hirsch, W.—(130), (134), Delusions, *159, 228, (264), (269); Amaurotic Idiocy....	*538, (559)	Krewer, L.—Landry's Paralysis.....	286
Hirschberg.—Fraenkel's Method in Tabes	501	Lancereux.—Alcohol	65
Hirtz and Lesne.—Landry's Paralysis	286	Landenheimer, R.—Diabetic Pseudo-Paresis	631
Hoche, A.—Pyramidal Tracts.....	215	Langdon	(677)
Hodge.—Friedreich's Disease..	70	Langley, J. N.—Regeneration Nerve Fibres	63
Hoffmann, J.—Hereditary Muscular Atrophy, 222; Muscular Dystrophy	893	Lannois.—Chorea	352
Holden, W. A.—Fluttering, 351; Amaurotic Family Idiocy, Eye Changes.....	*550	Lax and Müller.—Traumatic Disease of Spinal Cord.....	697
Howard, W. L.—Impulse to Drink	873	Lemoine, G.—Methylene Blue in Ataxia Pains	74
Jaboulay.—Cerebral Cysts.....	549	Le Noble, E.—Spinal Cord and Pernicious Anaemia	142
Jaccoud.—Acute Myelitis.....	702	Lépine, R.—Pyramidon.....	537
Jackson, H.	137	Lermoyez.—Recurrent Paralysis	64
Jacobi, Mary Putnam.—(200), (202), (820)		Leszynsky, W. M.—(30), (203), (264), (336), (483); Pachymeningitis.....	*609, (614)
Jacobson.—Capsular Tumor.....	568	Leuba, J.—Religious Phenomena, 146; Moral Imperative	630
Jacoby, G. W.—(53), (265); Myotonia	*508, (527)	Levet, M.—Crossed Paralysis.....	630
Jacquet, M. L.—Facial Paralysis	627	Lewis, W. B.—Cerebral Cortex, 62; Technic.....	347
Jelliffe, S. E.—227, 230, 359, 360, 504, 835, 901, 902, 904		Lloyd, J. H.—(45), (123), (127) 360, (622), (745), (752). Syringomyelia and Trauma, 871, 902	
Jemma.—Cure of Cerebro-Spinal Meningitis.....	150	Longues.—Congenital Amputation	67
Jemma and Bruno.—Lumbar Puncture	151	Luce, H.—Hemiplegia and Whooping Cough.....	626
Joukowski.—Frontal Lobes.....	347	Lugaro & Chiozzi.—Inanition.....	284
Juliusberger, O., and Meyer, E.—Ganglion Cell Pathology.....	626	Luisada, E., and Pacchioni, D.—Diphtheria Toxin.....	625
Jumon, L.—Glycosuria and Nervous Disease	887	Lyonnet and Bonne.—Hysterical Hemiplegia.....	355
Karplus, J. P.—Immobile Pupils in Hysteria and Epilepsy.....	895	Macalester, R. K.....	227
Keen, W. W.—Microcephaly, *105, Gasserian Ganglion. *800, (806)		MacLeod.—Morphine Habit Cured by Bromide.....	633
		Mairet.—Epilepsy	61
		Marie, P.—Is there Aran-Duchenne's Disease?	221

- | | PAGE. | | PAGE. |
|--------------------------------------------------------------------------------------------------------------------------------------------------|----------|------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|-------------|
| Marinesco.—Congenital Ampu-
tation, 67; Bulbar Paralysis,
68; Minute Anatomy Nerve
Cell, 138; Localization Motor
Nerves in Cord..... | 757 | Olmstead, I.—Cerebral Ataxia.*807 | |
| Martin.—Ocular Manifestations
in Tumor..... | 566 | Onuf, B. (60); Inhibition, 217,
(678), (823) | |
| Marty, A.—Treatment Sciatica.. | 293 | Onuf, B., and Collins, J.—Sym-
pathetic Nervous System.... | *661 |
| Mason, R. O.—Hypnotism..... | 175 | Oppenheim.—Dorsal Roots.... | 65 |
| McCasky, G. W.—Neurasthenia,
890 | | Ortner, N.—Collateral Hemi-
plegia | 763 |
| McConnell, J. W. (277), (618) | | Osler, W.—Vertigo..... | 68, (123) |
| McGrew.—Epilepsy | 177 | Ostankow, P.—Gastric Crisis... | 499 |
| McKerron.—Hemiplegia and
Whooping Cough..... | 61 | Overend, W.—Chorea Treat-
ment | 358 |
| Meirowitz, P. | 157, 229 | Packard, F. A.—(42), (43), (124),
(279), (342) | |
| Mendel, E.—Tabes and Multiple
Sclerosis and Trauma..... | 391 | Patrick, H.—(606); Spinal Irri-
tation, 746; Syringomyelia, *837;
Brain Tumor and Anemia. *882 | |
| Meyer, A.—Facial Paralysis,
284; Hereditary Ataxia, Path-
ology of..... | 494 | Patrizi, M. L.—Vascular Re-
flexes | 624 |
| Mills, C. K.—(50), (124), (125),
(210); Landry's Paralysis, *365,
(485), (558), (620), (804), (863) | | Patry, G.—Chorea..... | 353 |
| Mills, C. K., and Spiller, W. G.
—Landry's Paralysis | *365 | Pearce, F. S. (754), (756) | |
| Mills, W.—Cortical Localiza-
tion | 700 | Peli, G.—Prognathism..... | 867 |
| Minor, L.—Syringomyelia..... | 758 | Pergens, E.—Colored Lights on
Retina | 561 |
| Mitchell, J. K.—Hysteria and
Seborrhea nigricans, 354,
(485); Trigeminal Spasm,
*392, (615), (620), (622), (756) | | Pershing, H. T.—(32), Pressure
Neuritis | 288 |
| Mitchell, S. W., and Rhein, J.
W.—Chorea | 354 | Peterson, F.—(54), (57), (131),
(132), (133), (265), 297, Oph-
thalmoplegia chronica ex-
terna, *310, New Paths in
Psychiatry, *444, (483), Am-
autotic Idiocy..... | *529, (815) |
| Molliere and Perret.—Perimen-
ingitis | 463 | Peterson, F., and Langdon, W.
—Katatonia | 632 |
| Mongorer et Carrière.—Myelitis. | 759 | Petren, K.—Neuromata..... | 65 |
| Mongour.—Egotism | 629 | Pfister.—Technic | 757 |
| Monoco, L.—Optic Thalamus. | 350 | Pillsbury, W. B.—Appercep-
tion | 764 |
| Montgomery, T. H. (45) | | Pitres, A., and Carrière, G.—
Tabes and Arthropathies.... | 494 |
| Morasca.—Atrophy muscular.. | 221 | Potain.—Enteralgia | 826 |
| Morton, S. W.—Multiple Paral-
ysis and Measles..... | 288 | Prince, Morton.—Sexual Per-
version | *237 |
| Mouclaire, P.—Surgical Treat-
ment Facial Neuralgia..... | 358 | Probst, M.—Multiple Sclerosis. | 888 |
| Moyet.—Friedreich's Disease... | 70 | Pugnat, A. M. — Histology
Nerve Cells in Fatigue..... | 563 |
| Müller, L. R.—Tuberculosis of
Spinal Cord..... | 759 | Punton, J. (804) | |
| Mya, G.—Lumbar Puncture.... | 151 | Putnam, J. J.—(32), (487), (627),
(600); Post-Traumatic Psych-
oses, *769, (862), (869) | |
| Negro.—Chorea | 75 | Quartz, J. O.—Psychology of
Reading | 630 |
| Nonne, M.—Leukemia..... | 140 | Queyrat and Chretien.—Sy-
ringomyelia | 660 |
| Nouvatzke, H.—Cerebro-Spinal
Fluid | 138 | Raymond.—Amyotrophic Lat-
eral Sclerosis | 678 |
| Noyes, W. B. (129) | | Remlinger. — Landry's Par-
alysis | 286 |
| Ohlmacher, A. P.—Technic,
139; Pathology Epilepsy.... | 564 | | |

	PAGE.		PAGE.
Rhein, J. H. W.	(278)	Starr, M. A.—(33), 158, Friedreich's Ataxia.	*194, (599)
Riggs, C. E.—Bruce Microtome,	*865	Starr, M. A. and Weir, R. S.—Tumor of left Temporal Lobe	568
Riggs, E.—Friedreich's Disease	69	Sterling, J. W.—Tumor Meninges	566
Riley, W. H.—Tabes. Symptoms in	*679	Stewart, P.—Adductor Jerk.	219
Rivière, E.—Epicondylalgia.	290	Stieglitz, L.—(201), (202), (814)	
Rochet.—Paralytic Deformities.	634	Strauss, F.—Lumbar Puncture.	152
Rockwell, A. D.	(137)	Strümpell, A.—Chronic Inflammation Vertebral Column.	762
Rosenbaum.—Friedreich's Disease	71	Taylor, E. W.—(35), Family Periodic Paralysis.	*637, *719, (745)
Rosenthal, J., and Mendelssohn, M.—Reflexes	218	Taylor, W.—Neuroglia.	491
Ruffini, A.—Sensory nerve endings	282	Teichmüller.—Pernicious Anæmia	140
Sachs, B.—155, 158, (201), (270), (271), (272), 294, Multiple Sclerosis, *314, 336; Ophthalmoplegia, *452; Multiple Sclerosis, *464, (480), (483), (502), (555); Amaurotic Idiocy, 567, (592), (599), (815), (819), (823), (849)		Thayer	(341)
Sailer, J.—(280), 339, Hypertrophic Gliosis.	*402	Thomas, H. M.—(343), (345), Facial Paralysis.	*573, (592)
Savill.—Dermato-neuroses	895	Trepinski.—Tabes and Posterior Columns.	496
Schaffer, K.—Anterior Horn Cells in Tabes, 495; Tabes and Spinal Ganglia.	496	Tschistowitsch, T.—Healing of Cerebral Wounds	900
Schiff, A.—Lumbar Puncture.	629	Tucker, M. A.—Involuntary Movements	145
Schlesinger, H.—Abscess Spinal Cord	895	Upson, H. S.—Tumor of Oblongata	568
Schreiner, E.	(209)	Ursin, J.—Spinal Cord Changes in Brain Tumor.	563
Schlapp, M.—Monkey Cortex.	491	Valentine.—Neurasthenia.	75, 121
Schüle, V.—Katatonia.	632	Van Gehuchten, A.—Reflexes, 143, Reflex Movements, 218, Chromatolysis, 283, Muscular Rigidity	760
Schwartz, E.—Post-Anæsthetic Paralysis	760	Van Gieson, I.	(806)
Seguin, Edward Constant.	233	Villers.—Aran-Duchenne Atrophy	221
Siemerling, E.—Pupillary Reactions, 147; Muscular Atrophy	223	Vitzou.—Regeneration of Nerve Cells	63
Sinkler, W.—(206), 345, Epilepsy, *601, (608), (617), (620), (676), (744), (754), (756), (863), (870)		Vogel, K. M.	503, 504
Spiller, W. G.—Secondary Degenerations, *1, (50); Arrested Development, *81, (122), (127), (212), 231, (277), 339, 340, 345, 359; Landry's Paralysis, *365, (486), (527), (559), (592), (618), (620), (636), (756), (767); Gasserian Ganglion.	*800, (870)	Voisin, A.—Persecutory Delirium	560
Soukhanoff, S.—Polyneuritis.	284	von Sölder, F.—Constipation Psychoses	799
Spindler, F. N.—Touch	291	Voss, G. V.—Anæmia and Spinal Cord.	141
		Walton, G. L.	(613)
		Weber, L.—(600); Herpes Zoster	*868
		Welder, E.—Polyneuritis.	288
		Werbitzky, M.—Tabes.	570
		Werth.—Organotherapy	887
		Westphal, A.—Myelin Sheath Development	348
		Whitewell, J. R.—Neuroglia.	490
		Widal and Marinesco.—Bulbar Paralysis	68

	PAGE.		PAGE.
Wiener, A.—(332), (337). Ophthalmoplegia	*455	Worcester, W. L.—(33), Landry's Paralysis, *299, (656), (663); Nerve Fibre Regeneration	*698, (699)
Wilms.—Lumbar Puncture.....	152	Worotynski, B.—Secondary Degeneration	216
Witmer, A. F.—(40), (44), (617), (618)		Wullensoeber.—Central Softening of Cord	894
Wolfe, H. K.—Judgment of Size	562	Zaroubine, V.—Reflexes.....	220
Wolfe, J., and R.—Effects of Size on Judgments.....	357		
Wood.—Sixth Nerve paralysis..	899		

INDEX OF SUBJECTS.

Figures in parentheses () indicate discussions. Figures with asterisk indicate original articles and are accompanied with title. Figures unaccentuated, accompanied with title, indicate abstracts; without title, book reviews.

	PAGE.		PAGE
Abscess of Brain.....	(132), (276)	Arsenical Poisoning.....	(209)
Abscess in both Occipital Lobes		Arthropathies in Tabes....	494, 499
with Central Amaurosis.....	567	Arthropathy—Osteo-Hypertrophic	(341)
Abscess of Spinal Cord.....	895	Astasia Abasia.....	(206), (279)
Accessory Nerve Nucleus.....	346	Asymmetry in Face in Hemiplegia	628
Acoustic Paths.....	281	Ataxia and Methylene Blue....	74
Acromegaly and Hypertrophic Pulmonary Osteo-arthritis,		Ataxia—Cerebral	*807
(341)		Ataxia, Friedreich's, 69, 70, 71, 72,	*194
Adductor Jerk—Crossed.....	219	Ataxia—Hereditary Pathology.....	494
Aesthesiometer—A New.....	52	Ataxia—Locomotor. (See Tabes.)	
Akromegaly—A Case of.....	(40)	Ataxic Paraplegia.....	(754)
Akromegaly—The Brain and Spinal Cord from a Case of..	(42)	Athetosis and Infantile Cerebral Palsy	(814)
Alcoholic Drinks—Comparative Effect of.....	65	Atrophy—Muscular	*450
Alcoholic Paralysis with no Changes	285	Atrophy—Muscular and Thyroid	224
Amaurosis and Occipital Lobe Abscess	567	Atrophy Muscular—Progressive Spinal.....	221, 222, 223
Amaurotic Family Idiocy.....	*529, *538, *550, 567	Atrophy of Brain—Unilateral.....	(136)
American Neurological Association	31, 505	Blood—Alkalinity of, in Epilepsy	701
Amyotrophic Lateral Sclerosis.....	(134), (617), 678	Brain Tumor—Report of two Cases of.....	(332)
Anæmia and Brain Tumor....	*882	Brain Tumor and Anæmia....	*880
Anæmia—The Spinal Cord in Pernicious.....	140, 141, 142	Bulbar Paralysis—Descending. 68	
Anæsthesia in Tabes.....	*837	Bullet in Brain shown by Skiagraph	566
Anatomy — Cerebral Cortex, Structure of, First or Outermost Layer of.....	62	Capsule—Internal, Softening of, (275)	
Anatomy—Nerve Cells, New Formation of.....	63	Capsular Tumor.....	568
Anatomy — Regeneration of Pre-ganglionic and of Post-ganglionic Visceral Nerve Fibres	63	Cataleptoid Symptoms in Uræmia	570
Ankylosis of Spinal Column....	762	Cell Body—Functional Importance of	562
Aphasia	763, 897	Cells—Nerve, in Fatigue.....	563
Aphasia Sensory.....	824	Cells—Nerve, Structural Alterations	757
Aphasia—Subcortical Visual..	(337)	Cerebellar Tumor.....	(275)
Apperception	764	Cerebral Ataxia.....	*807
Aran-Duchenne—Muscular Atrophy	221	Cerebro-Spinal Fluid.....	138
Arsenic and Belladonna in Chorea	358	Cerebro-Spinal Fluid.....	138
		by Lumbar Puncture.....	150
		Chloæsis and Retinopapillitis.....	*874
		Chorea—Bacteriology of.....	564

	PAGE.		PAGE.
Chorea—Clinical Re-examination of Motor Symptoms....	354	Disseminated Insular Sclerosis,	(816)
Chorea—Huntington's....	352, *851	Dissociation in Pott's Disease,	*257
Chorea—Huntington's, Pathology and Morbid Anatomy of	(57)	Drink Impulse	873
Chorea—Lesions in.....	352	Dystrophy Muscular	893
Chorea—Paralytic	356	Echolalia	*20
Chorea—Pathology and Treatment of.....	74, 75	Electrical Stimulation of First Dorsal Root	65
Chorea—Treatment of.....	358	Electrical Therapeutics—New Device	(137)
Chorea—Variable or Polymorphous	353	Embolism—Choreic	354
Choreic Embolism.....	354	Encephalitis and Epilepsy.....	405
Chromatolysis	283	Encephalopathy Saturnine.....	77
Circulation—Intracranial	623	Enderteritic Myelopathy	892
Colloid Degeneration of Brain.....	565	Endogenous Fibres in the Cord	215
Conduction Paths for Reflexes.....	218	Enteralgia	826
Constipation Psychoses.....	799	Epicondylalgia	290
Cord Changes in Cerebro-Spinal Meningitis	565	Epilepsy and Encephalitis.....	401
Cord—Endogenous Fibres in the	215	Epilepsy—A New Sign of....	61
Cord Localization.....	757	Epilepsy—Blood in.....	701
Cord—Secondary Degeneration in	216	Epilepsy following Infantile Cerebral Palsy. Improvement after Craniotomy and Evacuation of a Subcortical Cyst	(30)
Cord—Spinal and Brain Tumor.....	563	Epilepsy—Hot Air as Treatment	489
Cord—Spinal and Tuberculosis.....	759	Epilepsy and Immobile Pupils.....	895
Cortex of Monkey.....	491	Epilepsy—Pathology of....	564, 758
Cortical Localization in Animals	700	Epilepsy—Remissions in.....	*601
Crossed Paralysis—Meningeal Hemorrhage	630	Epilepsy, Surgical treatment....	77
Cysts—Cerebral	549	Epilepsy—Trephining for....	(480)
Defective Brains—Five.....	(35)	Epileptics—Reflexes in.....	220
Deformities—Paralytic, Muscle Transplanting	634	Epileptics—Toxicity of Sweat.....	493
Degeneration — Colloid, of Brain	565	Ergotism and Local Asphyxia.....	629
Degeneration of Nervous System in Diphtheria.....	496	Eye—Motor Nerve Nuclei.....	346
Degeneration Secondary.....	216	Eyes—Muscular Sensibility of.....	493
Delirium Acute.....	72	Facial Hemiatrophy.....	224
Delusions and Gait.....	(278)	Facial Paralysis—Anatomical Findings in.....	284
Delusions — Psychical Mechanism of.....	*159	Facial Paralysis—Congenital.....	*573
Dementia Paralytica—Anterior Horn Cells in.....	493	Facial Paralysis—New Symptom of.....	289
Dermato Neuroses	895	Facial Paralysis in Typhoid Fever	889
Development of Myelin Sheaths	348	Family Optic Diseases.....	289
Diabetes and Insanity.....	146	Family Paroxysmal Paralysis.....	825
Diabetic Pseudo—Paresis.....	631	Fatigue—Histology Nerve Cells after	563
Diphtheria—Acute Degeneration in	496	Fever and Nerve Cells.....	351
Diphtheria Toxin on Nervous System	625	Fluttering	351
		Formaline as a Fixative.....	139
		Functional Importance of Cell Body	562
		Frenkel's Method of Treating Tabes	501

	PAGE.		PAGE.
Friedreich's Ataxia	69, 70, 71, 72 *194	lvanition and Nerve Cells.	283, 284
Frontal Lobes—Connections of	347	Infantile Cerebral Palsy and Athetosis	(814)
Frontal Lobe—Glioma of	*109	Inflammation—Chronic—Ver- tebral Column	762
Ganglion Gasserian	*800	Influenza and Neuritis	287
Ganglion Cell Pathology	626	Inhibition and Function of Py- ramidal Tracts	217
Gasserian Ganglion, Pathology of	*800	Insanity and Diabetes	146
Gasserian Ganglion Removal	*800	Insanity—Sedatives in	75
Gastric Crises	499	Insanity—Steps Toward	(38)
Glioma—Brain, Attempted Re- moval	568	Insomnia of Insanity—Treat- ment of	75
Glioma of the Frontal Lobe	109	Irido-plegia—Reflex Unilateral	(203)
Glioma of Medulla	569	Irritation—Spinal	746
Gliomatosis	*306	Judgments of Size	562
Glossitis—Hypertrophic Nodu- lar	*402	Katatonía	632
Glycosuria and Nervous Dis- ease	887	Katatoníc Melancholia	*747
Gonorrhœa and Polyneuritis	288	Kidneys in Cerebral Hemor- rhage and Cerebral Softening	(820)
Granules—Notes on	491	Knee Jerk Returned	615
Graves' Disease—Thyroid- ectomy in	766	Landry's Paralysis. 285, 286, *299,	365
Graves' Disease	(872)	Lameness, Intermittent	891
Gray Matter—Constitution of	561	Leukemia—Spinal Cord in	140
Hæmatomyelia, Traumatic	(821)	Lights on Retina—Action of Colored	561
Healing of Cerebral Wounds	900	Little's Disease	*81
Hemialgia	(273)	Localization Motor Nerves in Cord	757
Hemiatrophy Facial	224	Locomotor Ataxia (see Tabes).	
Hemiatrophy of Brain	(133)	Lumbar Puncture, 148, 150, 151, 152, 629	
Hematemesis in Neurasthenia	749	Measles and Multiple Paralysis	288
Hemiplegia and Edema	*189	Median Nerve—Suture of	(479)
Hemiplegia and Whooping Cough	626	Medulla—Glioma of	569
Hemiplegia in Whooping Cough	61	Medulla Oblongata—Tumor of	568
Hemiplegia—Collateral	763	Melancholia and Urea	631
Hemiplegia—Electricity in	634	Melancholia—Katatoníc	*747
Hemiplegia—Hysterical	355	Meningeal Hemorrhage and Crossed Paralysis	630
Hemiplegia—Infantile and Fa- cial Asymmetry	628	Meningeal Vascular Reflexes	624
Hemorrhage—Cerebral and Kidneys	(820)	Meningitis—Alcoholic and Brain Tumor	441
Hereditary Muscular Atrophy	222	Meningitis—Cerebro-Spinal Le- sions in Cord	565
Herpes and Facial Paralysis	627	Meningitis—Contribution to a Case of	(208)
Herpes Zoster	*868	Meningo-Myelitis	(752)
Hydrocephalus	(131)	Mental Phases of Tuberculosis	632
Hypnotism—Educational Uses of	174	Meralgia Paræsthetica	(756)
Hysteria and Toxic Tremor	(128)	Methylene Blue in Ataxia	74
Hysteria—Death in	19	Microcephaly—Operation in	*105
Hysteria—Seborrhœa Nigri- cans	354	Monkey Cortex	491
Hysteria and Immobile Pupils	895	Morphine Habit Cured by Bro- mide	633
Hysterical Contracture	355	Motor Reaction to Stimuli	765
Hysterical Hemiplegia	355		
Idiocy—Amaurotic Family, *529, *537, *549, 567			

	PAGE.		PAGE.
Movement of the Neuron.....	348	New York Neurological Society.....	53, 128, 200, 264, 332, 479.
Multiple Neuritis.....	594	Nuclei of Motor Nerves of Eye.....	346.
Multiple Neuritis—Recurrent.(343)		Ocular Manifestations in Intra-	
Multiple Sclerosis...*314, *464, 888		Cranial Tumor.....	566
Multiple Sclerosis and Func-		Ophthalmoplegia.....*452, *455.	
tional Tremor.....(620)		Ophthalmoplegia—A Case of	
Multiple Sclerosis and Trauma.....	391	Total and Complete Unilateral,	
Muscle Spindle.....	282	(45)	
Muscle Spindle in Pseudo-		Ophthalmoplegia Chronica Ex-	
Hypertrophy.....	219	terna.....	*310
Muscle Pathology.....	890	Optic Disease—Family.....	289
Muscular Atrophy.....	*450	Optic Thalamus—Physiology	
Muscular Atrophy—Hereditary.....	222	of.....	350
Muscular Atrophy—Progressive		Organotherapy in Chlorosis.....	887
Spinal.....	221, 222, 223	Pachymeningitis Hemorrhagica	
Muscular Dystrophy.....	893	Intern in Children.....	(54)
Muscular Rigidity.....	760	Pachymeningitis—Internal.....	*609.
Myelin Development.....	348	Paræsthetic Meralgia.....	(756)
Myelitis—Acute.....	702	Paræsthetic Neurosis—Nasal.....	(34)
Myelitis—Acute Dorso-Lum-		Paralyses—Post Anæsthetic.....	760
bar.....	759	Paralyses following Pasteur	
Myotonia.....	*508	Treatment.....	864
Myxœdema.....	*458	Paralysis—Periodic.....	*719
Nephralgia.....	199	Paralysis—Brown-Séquard.....	759
Nerve Cell Changes in Fevers.....	351	Paralysis—Bulbar, Descending.....	68
Nerve Cell—Minute Anatomy		Paralysis—Crossed and Meni-	
of.....	138	geal Hemorrhage.....	630
Nerve Cell Structure.....	214	Paralysis—Facial.....	289
Nerve Endings of Tactile		Paralysis—Facial, Congenital.....	*573
Hairs.....	281	Paralysis—Facial and Herpes.....	627
Nerve Fibre Regeneration.....	*698	Paralysis—Family Periodic.....	*637
Nerve, Sixth, Paralysis.....	899	Paralysis—Multiple and Mea-	
Nervous System—Apathy's		sles.....	288
Views on the Structure of.....	(45)	Paralysis—Paroxysmal, Family.....	825
Nervous System and Yellow		Paralysis, Sixth Nerve.....	899
Fever Bacillus.....	758	Paralysis—Transitory, of Car-	
Neuralgia—Facial Treatment,		diac Origin.....	356
Surgical.....	358	Paralytic Deformities.....	633, 634
Neurasthenia and Hematemesis		Paraplegia Ataxic.....	(754)
.....	749	Paraplegia—Hereditary Spas-	
Neurasthenia and Psycho-		modic.....	71
therapy.....	75, (121)	Paresis—General and Syphilis.....	147
Neurasthenia and Gastro-Intes-		Paresis—General, Anterior	
tinal Disease.....	890	Horn Cells in.....	493
Neuritis and Vaccination.....	(129)	Paresis—Pseudo and Diabetes.....	631
Neuritis—Arsenical, Following		Pasteur Treatment and Paral-	
Use of Fowler's solution.....	(209)	ysis.....	864
Neuritis—Multiple, Following		Pathology of Spinal Cord in	
Influenza.....	287	Traumatism.....	697
Neuritis of Fifth Nerve with		Pathology—Ganglion Cell.....	625
Herpes and Eczema.....	(277)	Pellot.....	600
Neuritis—Pressure.....	288	Perimeningitis—Primary Spi-	
Neuritis—Sciatic.....	291	nal.....	463
Neuritis—Syphilitic Multiple.....	*594	Persecutory Delirium Cured by	
Neuritis—Ulnar, Traumatic.....	(480)	Laparotomy.....	560
Neuroglia—Structure of.....	490, 491	Philadelphia Neurological So-	
Neurological Fragments.....	137	ciety.....	40, 122, 206, 273, 339, 485
Neuromata—General Multiple.....	65	Polyneuritis and Gonorrhœa.....	288

	PAGE.		PAGE.
Polyneuritis—Pathology	284	Scleroderma and Chronic Rheumatoid Arthritis	*703
Porencephalic Brain	(125)	Scleroses—Combined, in Pernicious Anæmia	142
Posterior Column Development and Tabes	496	Sclerosis—Disseminated	(816)
Post-Traumatic Psychoses....	*769	Secondary Degenerations following Cerebral Lesions....	*1
Prognathism	867	Sense of Position	493
Prognosis in Insanity	633	Sensibility of Eyes—Muscular ..	493
Psychology—		Sensory Nerve Endings in Muscles	282
Effects of Size on Judgment of Weight	397	Sexual Perversion	*237
Illusions—Study of	357	Skiagraph. See Röntgen Rays.	
Imaginations—Study of	357	Sleeping Sickness and its Bacillus	593
Psychology of Moral Imperative	630	Softening, Central, in Spinal Cord	894
Movements — Involuntary of Adults and Children	145	Spinal Column Anchylosis....	762
Psychology of Reading	630	Spinal Cord after Removal of Large Portions of the Limbs ..	67
Reaction Movements—Force and Rapidity of	145	Spinal Cord and Traumatism....	697
Religious Phenomena—Psychology of	146	Spinal Cord in Congenital Amputation of the Fingers	67
Psychoses—Due to Constipation	799	Spinal Cord in Leukemia	140
Psychoses—Post-Traumatic ..	*769	Spinal Cord in Pernicious Anæmia	140, 141, 142
Pseudo-Hypertrophy — Muscle Spindle in	219	Spinal Cord Abscess	895
Pseudo-Hypertrophic Paralysis.	220	Spinal Cord, Central, Softening in	894
Pseudo-Tabes in Diphtheria....	500	Spinal Ganglia in Tabes	496
Psychiatry—New Paths in....	*444	Spinal Ganglia of Mammals....	491
Psychic Anæsthesia—A Case of, (37)		Spinal Irritation	746
Pupils, Immobile, in Hysteria and Epilepsy	895	Structural Alterations in Nerve Cells	757
Pupillary Reactions in Mental Disease	147	Study and its Effects on the Nervous System	614
Pyramidal Tracts—Variations in,	215	Sympathetic Nerve Localization	*661
Pyramidon	537	Syphilis and Paretic Dementia..	147
Recurrent Paralyzes — Causes of	64	Syringomyelia	758
Reflex Movements	218	Syringomyelia	*837
Reflexes—Conduction Paths for ..	218	Syringomyelia—Localized ..	(202)
Reflexes—Exaggeration of, in Spastic and Hemiplegic....	143	Syringomyelia and Hemi-atrophy	660
Reflexes in Epileptics	220	Syringomyelia of Lumbar Cord ..	(488)
Reflexes in Syphilis	220	Syringomyelia and Trauma....	*871
Reflexes in Sciatica	220	Syringomyelic Dissociation in Pott's Disease	*257
Reflexes—Patellar in Tabes....	498	Tabes and Arthropathies....	494, 499
Regeneration of Nerve Fibres.*	698	Tabes and Embryonal Posterior Column Development....	496
Retina and Colored Lights....	561	Tabes and Floating Kidney....	498
Retinonapillitis and Chlorosis.*	874	Tabes and Multiple Sclerosis and Trauma	391
Rheumatoid Arthritis and Scleroderma	*703	Tabes and Spinal Ganglia Cells.	496
Röntgen Rays—Bullet in Brain, ..	566	Tabes and Taste and Smell....	499
Röntgen Rays in Surgery....	627	Tabes and Testicular Sensibility	499
Sciatica—Treatment	293		

	PAGE.		PAGE.
Tabes and the Re-education		Tickling and Laughing—Psy-	
Method	501	chology of.....	292
Tabes and Trunk Anæsthesia..*	837	Tinnitus with Hysteria.....	(618)
Tabes and Tuberculosis.....	(129)	Touch—After Sensations of...	291
Tabes—Anterior Horn Cells in	495	Trauma and Syringomyelia....	871
Tabes Arrested by Blindness,		Traumatism and Spinal Cord...	697
(122)		Treatment of Paralytic De-	
Tabes—Early Symptoms of...	499	formities	633, 634
Tabes — Pseudo, Following		Tremor—Graphic Study of....	562
Diphtheria	500	Tremor Simulating Multiple	
Tabes—Symptoms in.....	679	Sclerosis	(620)
Tabes—Treatment by Stretch-		Tremor, Toxic, and Hysteria in	
ing	500	a Male.....	(129)
Tabes — Treated with Sper-		Trigeminal Spasm	*392
minium-Poehl	570	Tuberculosis and Tabes.....	(129)
Tabes with Intercurrent Hemi-		Tuberculosis—Spinal Cord....	759
plegia, Returned Knee Jerk...	615	Tumor Brain and Changes in	
Tabes with Patellar Reflexes...	498	Spinal Cord.....	563
Tabes—Vomiting in	499	Tumor—Intracranial and Ocu-	
Taste and Smell in Tabes.....	499	lar Manifestations.....	566
Technic—		Tumor—Left Temporal Lobe...	568
Cerebral Development	(343)	Tumor of Inner Capsule.....	568
Chrome Silver Impregna-		Tumor of Meninges.....	566
tions	214	Tumor of Oblongata.....	568
Fixing Fluid	139	Tumor Cerebellar.....	(750), (752)
Formaline as a Fixative....	139	Tumors of Brain—Multiple..	(279)
Gold Staining of Nerve Fi-		Typhoid Fever and Isolated	
bres	281	Paralysis	889
Hardening Nervous System		Typhoid Fever and Suppuration	
in Situ	757	in Sella Turcica.....	(280)
Modification of Sublimate		Ulcer, Perforating—Cure of...	293
Method	347	Ulnar Nerve Injury.....	293
New Method of Staining Ner-		Unilateral Sweating and Flush-	
vous Tissue	139	ing	(278)
On the Nature of the Wiegert		Uræmia Cerebral with Cata-	
Pal Method	347	leptoid Symptoms.....	570
Present Methods of Prepara-		Vascular Diseases and Lame-	
tion Nervous System.....	623	ness	891
Temperature Sense.....	492	Vascular Reflexes in Meninges	624
Tetanus Lesions	626	Vertebral Column—Chronic In-	
Thomsen's Disease	(53), 608	flammation of.....	762
Thorns and Constitution of Gray		Vertigo and Equilibration....	*175
Matter	561	Vertigo and Ocular Defects....	68
Thyrocolloid	*458	Whooping Cough and Hemi-	
Thyroidectomy in Graves' Dis-		plegia	626
ease	766	Word Deafness—Autopsy.....	824
Thyroid Chlorosis	899	Wounds of Cerebrum, Healing	
Thyroid for Backward Children	881	of	900
Thyroid in Muscular Atrophy...	224	Yellow Fever Bacillus and Ner-	
Thyroidine	701	vous System.....	758
Tic Convulsif	(130), 356		

INDEX TO BOOK REVIEWS.

PAGE.	PAGE
A Manual of Legal Medicine, Justin Harold.....	635
Atlas of Legal Medicine, E. von Hoffmann	635
A Text Book of the Practice of Medicine, Jas. M. Anders....	503
An Epitome of the History of Medicine, L. Park.....	230
A Text Book on Mental Diseases, T. H. Kellogg.....	225
Beitrag zur Klinik der Rückenmarks- und Wirbeltumoren by Herman Schlesinger.....	767
Bulletin of the Ohio Hospital for Epileptics.....	297
Clinical Lectures in Mental Diseases, J. T. S. Clouston.....	158
Crime and Criminals, J. S. Christison	157
Die Bedeutung der Augenstörungen für die Diagnose der Hirn und Rückenmarks-Krankheiten, O. Schwartz....	359
Dysknesias Arsenicales, J. Moreira	227
Die Darstellung Krankhafter Geisteszustände in Shakespeare's Dramen, H. Laehr....	228
Die Geschwülste des Nerven Systems, von Ludwig Bruns....	901
Die Akromegalie, M. Sternberg	229
Eye Strain in Health and Disease, A. L. Ranney.....	157
Festschrift anlässlich des fünfzigjährigen Bestehens der Provinzial Irren Anstalt zu Nietleben bei Halle.....	903
Gehirnpathologie, I, Allgemeine Einleitung; II, Localisation; III, Gehirnblutungen; IV, Verstopfung der Hirnarterien, von Dr. C. v. Monakow	826
Hysteria and Certain Allied Conditions. Their Nature and Treatment with Special Reference to the Application of the Rest Cure. Massage, Electrical Therapy, Hypnotism, G. J. Preston.....	155
Hypnotism and its Application to Practical Medicine, H. G. Petersen	153
Les localizations des fonctions motrice de la moelle épinière, F. Sano.....	359
Leitfaden der Physiologischen Psychologie, von Pr. Dr. Th. Ziehen	835
Les affections nerveuses systematiques et la theorie des neurones, J. M. Gerest.....	359
Les hydrocephalies par Dr. Leon d'Astros	902
Les myelites syphilitiques, formes cliniques, et traitement, par le Dr. Gilles de la Tourette	901
L'hysterie aux XVII et XVIII siècles, Mme. G. Abricossoff....	360
Normale und pathologische Anatomie der Nervenzellen auf Grund der neueren Forschungen, A. Goldscheider and E. Flatau	636
Normal and Pathological Circulation of the Central Nervous System, W. Browning....	78
Psychologie de l'instinct sexuel par Dr. Joanny Roux.....	901
Psychology of the Emotions, Th. Ribot.....	360
Sulla opportunita ed efficacia della cura chirurgico-ginecologico nella neurosi isterica, Angellucci et Pierraccini....	227
System nerveux central, J. Dagonet	231
The Genesis and Dissolution of the Faculty of Speech, J. Collins	502
The Psychology of Suggestion, Boris Sidis.....	504
The Proceedings of the American Medico-Psychological Association	504
The Nervous System and its Diseases, Chas. K. Mills.....	294
Traumatic Injuries of the Brain and its Membranes, C. Phelps ..	78
Twentieth Century Practice....	80

THE
Journal
OF
Nervous and Mental Disease

Original Articles.

A CONTRIBUTION TO THE STUDY OF SECONDARY DEGENERATION FOLLOWING CEREBRAL LESIONS.¹

BY WILLIAM G. SPILLER, M.D.,

Professor of Diseases of the Nervous System in the Philadelphia Polyclinic; Pathologist to the Pennsylvania Training School for Feeble-Minded Children.

From the Wistar Institute of Anatomy and Biology.

This case and the following, 'K. K., have been obtained from the Pennsylvania Training School for Feeble-Minded Children.

The history of the boy, W. M., as far as known, is as follows:

He was born January 28th, 1882, and began to walk when about a year and a-half old. When he was little more than two years old, he "was frightened by a cat and was paralyzed," so that he had no use of the right upper limb. About this time he was noticed to be weak-minded. It seems probable that the vascular lesion, which caused the right hemiplegia, developed at this age. The boy learned to walk after braces had been applied to the right lower limb, but later these became unnecessary and were removed. He never used a crutch. Epileptic attacks first appeared when the child was ten years old, and at the age

¹One of two papers presented for membership in the American Neurological Association.

of thirteen these became more severe, and walking became impossible. In the convulsions the muscles of the entire body, as well as those of the head, were contracted. The mother states that the attacks were announced by screams, and that these were followed by partial unconsciousness. The boy never attended school, and was mentally not more developed than a child at the age of three. He spoke very indistinctly, but could be understood by his relatives, and seemed to understand what was said to him. He was left-handed, and the right upper limb was contracted and imperfectly developed. The labor is said to have been very difficult.

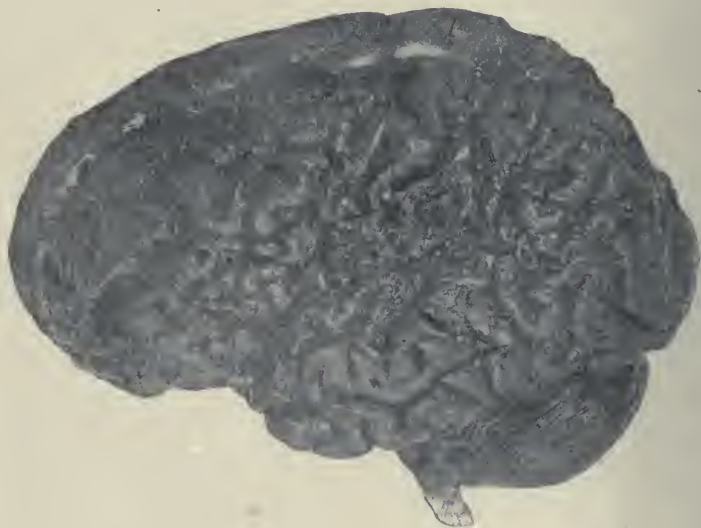


FIG. I.

The photograph represents very well the condition of the brain. The area nourished by the Sylvian artery was sclerotic. Only the cortex of the superior part of the central gyri was preserved, and yet the connection of this part with the internal capsule was destroyed. The foot of the third frontal gyrus was sclerotic, though to a less degree than the other parts of the Sylvian area. The first

temporal and supramarginal gyri and the insula were entirely destroyed, and the angular convolution was also affected. The entire left hemisphere was notably smaller than the right. The area of Broca and the centres for word-hearing and word-seeing were in the sclerotic region. The prefrontal lobe, the upper part of the central and parietal and the occipital and temporal lobes, in large part, were intact. These unaffected portions do not receive their blood-supply from the Sylvian artery. The brain, with the pons and oblongata, was hardened and cut in about nine hundred serial sections. The terminology employed in this description is chiefly that used by Dejerine in his *Anatomie des Centres Nerveux*.

The large pyramidal cells (*Riesenzellen*) are absent in the upper part of the central gyri in sections taken from a portion which externally appeared normal, and indeed the smaller cells are not as numerous as in a normal brain, and are irregular and very imperfect.

Sections Nos. 875 to 860 through the upper part of the tail of the caudate nucleus show that the sclerosis involves the posterior limb of the internal capsule so completely that only a very few horizontal medullated fibres may be found within it. The inferior longitudinal fasciculus, the optic radiation, and the tapetum are interrupted by the primary lesion. A narrow band of fibres near the tail of the caudate nucleus, and a larger band more posteriorly on the lateral side of the ventricle are left. The tail of the caudate nucleus has not been destroyed, and contains many fine black points by Weigert's stain, representing bundles of fibres cut transversely. Within the posterior part of the head of the caudate nucleus (the anterior part is not in the sections) there are also many of these points. The anterior limb of the internal capsule at this high level shows a slight degeneration, though it is intact at lower levels. The sections contain the uppermost part of the thalamus, which is very small. It has been impossible to keep the sections from the two hemispheres in the same plane, as the left half of the brain was so contracted. The cortex consists of sclerotic tissue.

Sections Nos. 846 to 833:—The few fibres present in the posterior limb of the internal capsule have almost entirely a horizontal course, and pass into the external nucleus of the thalamus. The anterior limb of the internal capsule is nearly normal, though smaller than that of the right side. The fibres

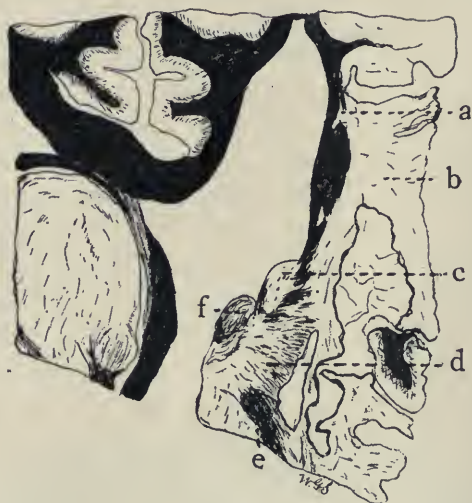


FIG. II.

Section 860. (a) Optic radiation and inferior longitudinal fasciculus; (b) degenerated cortex; (c) tail of the caudate nucleus; (d) posterior limb of the internal capsule; (e) anterior limb of the internal capsule; (f) thalamus.

present in the knee of the internal capsule have a horizontal course and seem to connect the anterior part of the putamen with the thalamus, though some, evidently, belong more directly to the anterior limb of the capsule. The extreme anterior end of the putamen is preserved, and contains a number of horizontal medullated fibres. All the middle and posterior part of the putamen is involved in the primary lesion. The posterior part of the head of the caudate nucleus (the only part in the sections) appears normal. The anterior nucleus of the thalamus is smaller than that of the right side, and contains fewer medullated fibres. The stratum zonale is preserved, as are also the laminæ medullares of the thalamus. The external nucleus of the thalamus is very small, but contains many medullated fibres. At this level the posterior part of the thalamus receives a certain number of fibres from the optic radiation. The tail of the caudate nucleus is intact. Only the anterior part of the external capsule is preserved. There are a few masses of gray matter belonging to the posterior part of the putamen. These give origin to numerous horizontal fibres which pass into the external nucleus of the thalamus; some, however, pass only part way through the sclerotic tissue of the posterior limb of the internal capsule.

Sections Nos. 827 to 816:—As the perpendicular fibres of the posterior limb of the internal capsule are destroyed, except a very few, it is important to notice the horizontal fibres which connect the lenticula with the external nucleus of the thalamus. The second segment of the lenticula is now visible and contains many medullated fibres. The horizontal fibres of the posterior limb of the internal capsule are somewhat more numerous in the posterior part of the limb. The inferior longitudinal fasciculus and the optic radiation contain more fibres than in sections from higher levels. The anterior nucleus of the thalamus has become much smaller. The internal nucleus is distinct, but is smaller than that of the right side. The *tænia thalami* is well preserved. The anterior limb of the internal capsule is intact, and some fibres in the knee of the capsule are preserved. The inferior longitudinal bundle, optic radiation and tapetum at some little distance from the tail of the caudate nucleus are much degenerated, but as there are many fibres in the retrolenticular segment of the internal capsule, they must represent fibres which ascend in their course forward. A large part of the putamen and external capsule are destroyed. The habenula is well formed.



FIG. III.

Section 812. (a) Optic radiation and inferior longitudinal fasciculus; (b) posterior limb of the internal capsule (degenerated); (c) posterior part of the putamen destroyed by the primary lesion; (d) anterior part of the putamen (normal).

Sections Nos. 812 to 800:—Only the posterior part of the putamen is destroyed. The second segment of the lenticula is well formed, and contains many medullated fibres. The first segment also appears normal. The posterior limb of the internal capsule is much contracted. The internal nucleus of the thalamus is distinctly separated from the external by the lamina medullaris interna. The pulvinar begins to be prominent. The lamina medullaris externa and the zona reticulata are distinct. The laminae of the lenticula are also well formed. The bundle of Vicq d'Azyr on the left side is a trifle smaller and less deeply stained. The anterior pillars of the fornix are the same on two sides. The tænia semicircularis is of good size and well stained. The fibres of the inferior longitudinal bundle, optic radiation and tapetum are only interrupted at one portion of their course. The zone of Wernicke is deeply stained and appears normal, as only the uppermost part of the optic radiation was cut. The fasciculus of Türck begins to appear. The external capsule now forms an unbroken band, but the fibres are not numerous. There are medullated perpendicular fibres present in the extreme anterior part of the posterior limb of the internal capsule.

Sections Nos. 799 to 792:—The posterior commissure is normal. The putamen is no longer destroyed in its posterior part. The pulvinar is not quite as large as that of the right side at higher levels, and probably this is the result of destruction of the superior fibres belonging to the optic radiation. Fibres may be noticed passing toward the anterior and outer part of the posterior limb of the internal capsule. These are coarser than the fibres which enter the thalamus directly. Some of these coarser fibres appear to pass through the anterior part of the posterior limb of the internal capsule into the thalamus, for though it is impossible to trace them in their passage through the capsule, there are fibres on the outer side of the bundle of Vicq d'Azyr which appear to be the continuation of these. Some of these fibres, however, seem to remain in the anterior part of the posterior limb of the internal capsule, and from this level medullated fibres may be traced in this part into the median bundle of the peduncle.

Sections 784 to 773:—The fasciculus retroflexus of Meynert, arising in the habenula, is well colored. The horizontal fibres connecting the thalamus with the lenticula are more numerous than in sections from higher levels. The fasciculus of Vicq d'Azyr is well stained, and is about the size of that on the right side. The thalamic fasciculus of Forel (*Markfeld H1*) is distinct, as is also the lenticular fasciculus of Forel (*Markfeld H2*). These do not appear to be atrophied, or if so, they are to a very slight degree. The median nucleus of Luys has

nearly the normal size, but the semilunar nucleus of Flechsig, so called by Dejerine, (*Schalenförmiger Körper*) is very small. Fibres from the lamina medullaris interna of the lenticula begin now to pass through the internal capsule. The pulvinar is of good size, and the zone of Wernicke is intensely stained. The fasciculus of Türck is very distinct. The left anterior pillar of the fornix is fully as large as the right. The left ansa lenticularis at this level is smaller than the right, but this is chiefly the result of a difference in the level of the two sides. The zona incerta is small.

Sections Nos. 755 to 743:—The ansa lenticularis is a little smaller on the left side. The internal geniculate body appears in its most superficial part, and is much atrophied. The upper portion of the external geniculate body is also visible, and the peculiar divisions of this body are very distinct. The peduncle of the anterior quadrigeminal body is intact. The peduncle of the posterior does not contain the normal number of fibres. The median side of the capsule of the nucleus ruber, *i. e.*, the *Haubenstrahlung* in the sense of v. Monakow,—contains fibres from the ansa lenticularis (Dejerine), and no difference in the size of this median portion in the two hemispheres is observable. The left nucleus ruber is much smaller than the right, but the level on the two sides is not the same. The anterior com-

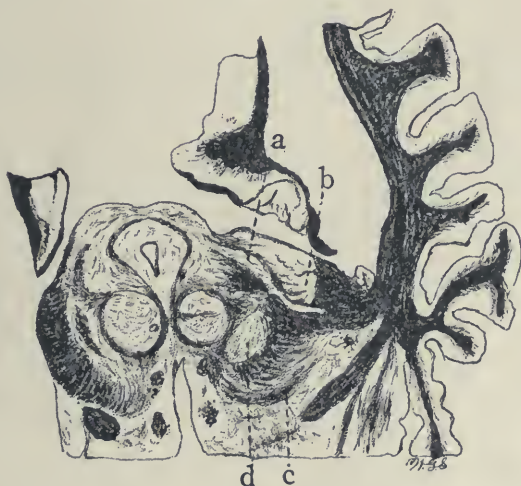


FIG. IV.

Section 736. (a) Internal geniculate body (partly atrophied); (b) external geniculate body; (c) ansa lenticularis; (d) corpus subthalamicum.

missure is small. The posterior longitudinal bundles are the same on the two sides. The left corpus subthalamicum may be a little smaller than the right in its greatest development, but there is no great difference between the two. In Mahaim's² case this body was much degenerated, but the destruction of the putamen seems to have been greater.

Sections Nos. 736 to 724:—The peduncle of the posterior corpus quadrigeminum contains quite a number of fibres at this level, though not as many as the corresponding bundle on the right side at higher levels. The ansa lenticularis is well formed at this level and cannot be regarded as degenerated to any noteworthy degree. The anterior part of the posterior limb of the internal capsule in all these sections contains medullated fibres. The commissure of Meynert is normal, and fibres of this band may be seen entering the posterior part of the internal capsule. These fibres are of fine calibre.



FIG. V.

Section 676. (a) Lateral bundle of the peduncle; (b) internal bundle of the peduncle.

Sections 714 to 661:—The left nucleus ruber is about equal in size to the right, though the latter in its greatest development is larger. The peduncle of the lateral mammillary body is normal, as is also the tubercle itself. Many horizontal fibres may be seen passing from the substantia nigra into the crusta, which is not as yet detached from the rest of the brain. The area which in higher levels is much degenerated, and which corresponds to the portion occupied by the motor fibres, contains many horizontal fibres. The commissure of Forel and the optic nerve are normal. The two mammillary bodies are about the same in size. The substantia nigra of the left side apparently contains as many cells and fine fibres as that in the right peduncle, but as the left crus is much smaller than the right the total number of cells in the substantia nigra is probably also less. The median portion of the crusta is well colored, as is also the lateral, which is about one-fifth of the width of

²Mahaim: *Archiv für Psychiatrie*, vol. xxv.

the normal crista. This lateral bundle contains no visible degenerated fibres. The fibres passing from the nucleus ruber to form the anterior cerebellar peduncle of the right side are apparently as numerous as on the left side. The lateral lemniscus is normal, and may be seen passing toward the nucleus of the posterior corpus quadrigeminum. This nucleus is of good size.

Below section 661 it is not worth while to give the numbers, as the changes are not sufficiently numerous to justify such a procedure. There is no apparent difference in the number of the fibres constituting the middle cerebellar peduncles, but the left half of the pons is much narrower than the right. The right anterior cerebellar peduncle is a little smaller than the left. There are only a few perpendicular medullated fibres enclosed by the left middle cerebellar peduncle. The nuclei of the pontine gray matter seem to be equally numerous on the two sides, but there are probably not as many of these on the left side *in toto*. The left median lemniscus is smaller than the right. There are many cells in both sensory and motor nuclei of the fifth nerve on each side, and the spinal roots of the two fifth nerves seem to be of equal size. The nuclei of the sixth nerves are normal. The cells of the formatio reticularis appear equally numerous on the two sides. The ventral acoustic nuclei and the acoustic tubercles are well formed on both sides. The left interolivary layer is one-third smaller than the right, and the fibres appear to be of smaller calibre, and yet this is a point most difficult to decide, although the method of coloration by the carmine *en masse* has been employed, and the result is excellent. The left pyramid is entirely degenerated. The right pyramid does not appear extraordinarily large, although it is of full size. The nuclei of Burdach and Goll are smaller on the right side, and contain fewer cells. At the motor decussation there are fibres which appear to penetrate the gray matter to enter the lateral column on the side of the normal pyramid. The cranial nerves and nuclei all appear to be normal.

A history of a fright, which was supposed to have been the cause of the paralysis, is given in this case. It is difficult to picture any morbid process which could be produced in this way. A much more probable explanation is that the fright occurred simultaneously with the occlusion of the Sylvian artery. In attempting to remove this artery from the hardened brain it broke near the sclerotic area. Nothing pathological could be observed in the piece thus obtained. The cortical branches of the artery could not

be distinguished from the sclerotic tissue. It is impossible to state whether the occlusion was the result of a thrombus or embolus. Most physicians would probably think of the latter cause, though Gowers³ believes that thrombosis *in situ* is more common in such cases as this. Taylor,⁴ however, reports a case in which embolism of the left middle cerebral artery was found in a child of five. The feeble intellect of W. M. was probably the result of this sclerosis, although the prefrontal lobe was well developed. The intellect may thus be of low grade when the greater part of the prefrontal lobe is intact, but considerable destruction of cortical tissue in some cases has not made the patient feeble-minded (v. Monakow's⁵ case, No. 3). There may be a difference in this respect in proportion to the development of the brain at the time the lesion occurs. Probably a congenital lesion in some cases may not produce the same symptoms as one which occurs when the child is two or three years old; and, therefore, at an age when the brain has attained a higher degree of development, and the nerve cells are less likely to assume abnormal functions.

The motor fibres of the left cerebral hemisphere were totally destroyed, and yet this boy was able to walk without a crutch, although in an imperfect manner. He had no use of the right upper limb. The conviction is forced upon us that the motor impulses to the right lower limb were transmitted through the pyramidal fibres from the right cerebral hemisphere. This preservation of bilateral motion with the absence of one pyramid is well shown in another case from the Elwyn institution. The pyramid on the same side as the normal hemisphere in this case is unusually large, and there is entire absence of the left pyramid. The case is one of porencephaly of the left lower

³Gowers: Diseases of the Nervous System, vol. ii., English edition, p. 461.

⁴Taylor: British Medical Journal, 1880, vol. ii.

⁵V. Monakow: Archiv für Psychiatrie, vol. xxvii.

Rolandic region, and a picture of this brain was given in the January, 1897, number of the *JOURNAL OF NERVOUS AND MENTAL DISEASE*. This boy also was able to walk everywhere, although he limped, and the only motor fibres present were from the right pyramid. In the case of W. M., the right pyramid is not as large as in the case of porencephaly. It is probable that in the first case the lesion was post-natal, and in the second ante-natal, which accounts for the better compensatory development of fibres in the second case. A case in which the innervation of both sides of the body was probably from one cerebral hemisphere, has been reported by v. Monakow,⁶ and the same explanation as above has been given. In Mahaim's⁷ case, the lesion was in the Sylvian region, and involved the supramarginal, the first temporal gyrus, the insula, the claustrum, almost all the putamen, the head of the caudate nucleus, and the fibres from the central gyri. The hemiplegia developed at the age of nine months. Although the fibres from the right motor cortex were destroyed, the gait of the patient was not strikingly abnormal, and spastic phenomena were not present in the left lower limb. The left upper limb was contracted, but still of service. Mahaim says this case is an evidence of the connection of each pyramid with both sides of the cord, and this bilateral innervation is more perfect for the lower limbs than for the upper. Dejerine and Thomas⁸ have reported a case similar to these. In one of v. Monakow's cases of infantile cerebral paralysis contracture of the upper limb, with shortening and diminution in the circumference of the forearm, was noticed, and yet the pyramid containing the fibres for this side of the body was only moderately degenerated. The hemiplegia developed at the early age of six months, and, therefore, at a period when compensation as a rule is more perfect. V. Mona-

⁶V. Monakow: *Archiv für Psychiatrie*, vol. xxvii, p. 408.

⁷Mahaim: *Archiv für Psychiatrie*, vol. xxv.

⁸Dejerine and Thomas: *Archives de Physiologie*, 1896.

now says this case shows that a partial interruption of the motor fibres may cause a considerable hemiatrophy with contracture. On the other hand, occasionally in cases of total pyramidal degeneration the hemiatrophy and contracture may be less, as in Mahaim's case, and consequently the hemiatrophy is not directly proportional to the pyramidal degeneration, but is dependent on other unknown factors. Zacher⁹ also has reported a case of partial degeneration of the pyramidal tract without evidences of paralysis during life. It is a curious coincidence that the epileptic attacks developed in the boy, W. M., at the same age as in the patient reported by v. Monakow as Case 3. In both cases the convulsions began at the age of ten and were general. This is additional proof that both sides of the body were innervated from one hemisphere. It is well known that extirpation of motor centres usually causes a cessation of epileptic movements in the corresponding limb. It seems probable that these bilateral convulsions were made possible by the formation of a lesion at an early age. The nervous system can adapt itself much better to altered circumstances if destruction of tissue occurs before the nerve cells and fibres are fully formed, and it would seem that even additional fibres may develop (cases of Mahaim, v. Monakow, Dejerine and Thomas, Adolf Meyer¹⁰ and Spiller).

W. M. spoke very indistinctly, but he was able to make himself understood, and appeared to understand any simple remark made to him. This is very interesting as the fibres from Broca's region were certainly very much damaged, and Freud's speech zone was sclerosed. Such a lesion in the adult causes both motor and sensory aphasia. V. Monakow reports a similar condition in his Case 2, and other cases are known in the literature.

⁹Zacher: *Archiv für Psychiatrie*, vol. xxvii.; see also vol. xix.

¹⁰Meyer: *Pathological Report of the Illinois Eastern Hospital for the Insane*.

Many years ago v. Monakow showed that the internal geniculate body degenerates after lesions of the temporal lobe. According to Zacher,¹¹ Wernicke claimed that there is direct connection between this nucleus and the first temporal gyrus and the insula, and Zacher, from a study of his cases, is inclined to accept this statement, although he thinks the second temporal gyrus is probably also connected with this nucleus. This connection with the temporal lobe has been also observed by others. In v. Monakow's paper, to which reference must repeatedly be made, the internal geniculate body in Case 2 was degenerated from a lesion in the first temporal gyrus, the operculum, the insula, the third frontal convolution, and the putamen. This body was also smaller on the side of the lesion in Case 3. In my case this body is much atrophied, though not entirely absent, and destruction of the cortex of the hemisphere is extensive. The first temporal gyrus is entirely destroyed, and it is with this that the internal geniculate body is chiefly connected (v. Monakow). There is also decrease in the size of the peduncle of the posterior corpus quadrigeminum, as was seen also in v. Monakow's Cases 2 and 3. The nucleus of this quadrate body in the case W. M. is of good size. After extensive lesions of the operculum and temporal lobe the atrophy of the peduncle of the posterior corpus quadrigeminum is only of moderate intensity (v. Monakow). This is because the cortical neuron begins in the internal geniculate body. The case of W. M. confirms this statement.

Mahaim and v. Monakow in his Case 2 report degeneration of the ansa lenticularis, but the destruction of the putamen in both these cases was evidently greater than in my case. The ansa lenticularis on the left side of the brain of W. M. is very nearly as well developed as on the right, and this is probably to be attributed to the fact that

¹¹Zacher: *Archiv für Psychiatrie*, vol. xxii.

the destruction of the lenticula was limited to the posterior and superior part of the putamen, and did not extend very far downward. In Mahaim's case the ansa lenticularis was not as much degenerated as in v. Monakow's, as the latter states. The lenticular fasciculus of Forel, and the lenticular laminæ in v. Monakow's Case 2 contained fewer fibres. These parts are quite well formed in the case W. M., and the median portion of the tegmental radiation (*Haubenstrahlung*) on the left side contains as many fibres as on the right. According to Dejerine the ansa lenticularis contributes fibres to the tegmental radiation. In the sense of v. Monakow (l. c., p. 113), the tegmental radiation includes all the fibres immediately surrounding the nucleus ruber. It seems probable that the ansa lenticularis arises chiefly from the lower part of the lenticula.

Zacher, Mahaim, and v. Monakow report degeneration of the nucleus ruber on the side of the primary lesion. In the case W. M. the left nucleus ruber is a little smaller than the right, but contains proportionally as many medullated fibres. The substantia nigra has been reported as atrophied in several cases of cerebral lesions. In the case W. M. this is not very evident. The substantia nigra is smaller, as the whole cerebral peduncle is smaller, but proportionally speaking it seems to contain a normal number of cells and fibres. According to v. Monakow this part is probably connected with the third frontal gyrus and anterior part of the insula and operculum. Edinger¹² states that fibres of the stratum intermedium come from the lenticula. V. Bechterew¹³ says that the cells of the substantia nigra do not degenerate after lesions of the cortex and internal capsule. This substance was degenerated in Witkowski's¹⁴ case and the lesion involved the lenticula with other parts of the brain.

¹²Edinger: *Vorlesungen über den Bau der nervösen Centralorgane*. Fifth edition, pp. 288, 257.

¹³V. Bechterew: *Archiv für Psychiatrie*, vol. xix.

¹⁴Witkowski: *Archiv für Psychiatrie*, vol. xiv.

In Case 2 of v. Monakow's paper the cerebral peduncle was intensely degenerated on the median and lateral borders. These are intact in the case of W. M. Dejerine¹⁵ believes that the median bundle of the peduncle comes from the Rolandic operculum and adjacent part of the frontal operculum. Zacher (l. c.) believes this portion of the crusta is chiefly connected with the insula. Flechsig and Wernicke think this bundle comes from the frontal lobe (quoted by v. Monakow). V. Monakow is in favor of the latter view after a study of his Case 2. In the case W. M. the most internal portion of the inner bundle of the crusta is well stained and may be traced from quite high levels of the internal capsule in the anterior part of the posterior limb. The operculum is degenerated. This seems to indicate that the origin of a portion of this bundle is anterior to the Rolandic operculum. In Mahaim's case (l. c.) this median bundle was degenerated, but the head of the caudate nucleus was destroyed by the primary lesion, and it is probable that the anterior limb of the internal capsule was also affected.

The degeneration of the lateral bundle of the crusta in v. Monakow's Case 2 is noteworthy, as the second and third temporal gyri were not degenerated. It may be that the fasciculus of Türck was cut internally to the cortex of the temporal lobe in its passage to the posterior part of the posterior limb of the internal capsule. In Mahaim's case (l. c.) the external fourth of the cerebral peduncle contained normal fibres, and the first temporal gyrus was involved in the primary lesion. V. Bechterew¹⁶ in 1888 reached the conclusion that the lateral bundle of the cerebral peduncle arises in the temporal and basal part of the occipital lobe. Zacher (l. c.) also places the origin of these fibres in the occipital and temporal lobes. Dejerine (l. c.) has shown that the origin of this bundle is in the temporal

¹⁵Dejerine: *Anatomie des Centres Nerveux*, vol. i, p. 602.

¹⁶V. Bechterew: *Archiv für Psychiatrie*, vol. xix.

lobe alone, and chiefly from the second and third convolutions. Kam¹⁷ also believes that these fibres come only from the temporal lobe. Mills and Spiller¹⁸ have shown that probably none of these fibres arise in the first temporal gyrus. This case, W. M., strengthens this latter view. The first temporal gyrus was entirely destroyed at an early age, and secondary degeneration elsewhere is distinct, and yet no indication of degeneration is found in the lateral bundle of the peduncle.

The anterior and internal nuclei of the thalamus in Case 3 of v. Monakow's paper were almost of normal size. The central gyri with the operculum, the anterior part of the supramarginal gyrus, and the first temporal were destroyed by porencephaly. Only the frontal lobe and portions of the temporal lobe remained in connection with the internal capsule. This case in many respects is similar to mine. The left anterior nucleus in my case is only a little smaller than the right, and the internal nucleus is not nearly as much altered as the external (lateral). The lateral nucleus is greatly degenerated, as it was also in v. Monakow's Cases 2 and 3, in which also the ventral nuclei, as he calls them, and the posterior nucleus were degenerated. In Mahaim's case the anterior tubercle was the only thalamic nucleus intact, though the pulvinar was only a little smaller. From eleven human brains and from a number of brains of lower animals, in which experimental lesions were produced, v. Monakow concludes that every portion of the thalamus is in connection with some part of the cerebral cortex. Other cases in the literature support his views. This is contrary to the opinion held by Flechsig as regards his association centres. According to v. Monakow, a thalamic nucleus may not degenerate if some of the fibres passing to it are intact. Fibres from the first and second parietal gyri are in connection with

¹⁷Kam: *Archiv für Psychiatrie*, vol. xxvii.

¹⁸Mills and Spiller: *Journal of Nervous and Mental Disease*, 1896.

the frontal and median portions of the pulvinar (v. Monakow). This probably explains the slight diminution in the size of the left pulvinar, as compared with the right, in the case of W. M. The ventral nuclei of the thalamus are supposed to be chiefly connected with the operculum, the central and the supramarginal gyri. The semilunar nucleus of Flechsig (Dejerine) is identical with v. Monakow's vent. b. nucleus. The ventral nuclei are the groups of cells situated in the ventral portion of the thalamus. In the case W. M. these cortical areas are degenerated, and the area occupied by the ventral nuclei is small. The internal nucleus probably receives its fibres from the second and third frontal gyri and the anterior part of the insula. This explains the moderate degeneration in the case of W. M. By lateral nucleus v. Monakow means only the dorsal part of the external nucleus. It is probable that this nucleus derives its fibres from the central gyri (operculum), the superior parietal, the anterior part of the supramarginal, the angular, the posterior part of the frontal gyri, and the temporal lobe. We have thus a satisfactory explanation for the great degeneration of the lateral nucleus in the case of W. M. It is certain, says v. Monakow, that fibres to the lateral nucleus come chiefly from the central gyri. The anterior tubercle of the thalamus probably receives fibres from the median portion of the first frontal, the paracentral lobe and gyrus fornicatus. All these parts were outside the sclerotic area, and yet as some of the projection fibres were doubtless cut, we have an explanation for the slight degeneration of the anterior tubercle in the case of W. M.

The preservation of the external geniculate body, of the peduncle of the anterior quadrigeminal body, and of this body itself, are explained by the normal condition of the optic cortex and the preservation of the optic radiation, except in its most superior part.

The corpus mammillare receives fibres from the uncus,

cornu ammonis and surrounding tissue, through the fornix. None of these parts were degenerated in the case of W. M. In another case, which has only been examined macroscopically, and which I hope to be able to report later, more in detail, the occipital lobe is destroyed and the cornu ammonis is, to all appearances, much affected by the primary lesion. The corpus mammillare on the same side is only half as large as the corresponding body on the normal side of the brain.

The corpus subthalamicum is not closely connected with the cerebral cortex, but it is with the caudate nucleus and anterior part of the putamen (v. Monakow). Only the posterior and superior part of the putamen was destroyed in the case of W. M. The normal appearance, or at least very slight diminution in the size of the subthalamie body, may be easily explained.

The fibres of the nucleus ruber have been found atrophied after lesions of the cortex of the operculum, the second parietal gyrus and possibly the temporal lobe (v. Monakow). This nucleus is not much smaller on the left side than on the right in the case W. M.

The sensory nucleus of the fifth nerve on the side opposite to the cerebral lesion is not degenerated; the case therefore supports the statements of Mahaim (l. c.), but is contrary to those of Hösel.¹⁹ The latter asserts that the sensory nucleus of the fifth nerve is in direct connection with the central gyri of the opposite side. It is well known that Hösel found the contralateral sensory nucleus of the fifth nerve atrophied, after a lesion of the central gyri. The right anterior cerebellar peduncle is a little smaller than the left. Unfortunately, a portion of the tissue was lost just at this part by the sections which were made for the purpose of hardening. Hösel (l. c.) found the anterior cerebellar peduncle on the side opposite the lesion one-third less in size, and he believed his case

¹⁹Hösel: *Archiv für Psychiatrie*, vol. xxiv., xxv.

showed that there is a connection of this with the central lobe. Edinger believes the connection is indirectly with the parietal lobe. According to Hösel, Meynert also thought that there is a connection of the nucleus ruber with the parietal lobe. V. Monakow also reports atrophy of the contralateral anterior cerebellar peduncle.

In sections taken from the extreme upper part of the ascending frontal convolution which externally appeared to be normal, as may be seen in the picture, search was made for the giant pyramidal cells (Betzi). These cannot be found. Mahaim states that Moeli and Henschen as well as he himself have noted the absence of these cells in cerebral lesions in man.

LA MORT DANS L'HYSTERIE (Death in Hysteria). (Journ. de Méd. et Chir. prat., Aug. 25th, '96.)

Although the manifestations of hysteria are usually considered to be of a benignant character, cases have been observed in which death, due to various conditions, has occurred suddenly. Dr. Le Fournier, in a thesis, has collected a number of illustrative cases, and points to the dangers connected with spasm of the glottis, and anorexia. Thus several cases are reported in which tracheotomy was resorted to, in order to prevent asphyxia. A child, six years old, in imminent danger of being asphyxiated, the attack lasting one hour, was saved by applying the faradic current. Another case was that of a girl, 20 years old, subject to laryngeal spasms, who was revived by artificial respiration, but who died shortly afterwards in a similar attack. Hysterical anorexia ends fatally quite often. After a certain duration the patients fall into a semi-comatose condition, become sleepless, greatly emaciated, and bedridden. If fed artificially in this state the patients continue to lose flesh, the stomach being unable to perform its functions. Death may also occur from incessant vomiting, as for instance: A patient, who was subject to attacks of hysterical vomiting, after taking her evening meal as usual, went to bed and died during the night. The autopsy, made thirty-six hours after death, revealed nothing abnormal of note, and the stomach contained four ounces of food. Occurrence of sudden death in hysterical subjects has further been observed to be due to attacks of angina pectoris, and supervening upon abdominal operations, especial vaginal hysterectomy. MACALESTER.

SOME NOTES ON ECHOLALIA,¹ WITH THE REPORT OF AN EXTRAORDINARY CASE.²

BY MARTIN W. BARR, M.D.,

Chief Physician Pennsylvania Training School for Feeble-Minded Children,
Elwyn, Pa.

WITHIN the last decade contributions to literature relating to the various forms of cerebral speech disturbance have been more or less voluminous, but they have been devoted almost exclusively to those most frequently met with, and the rarer one of echolalia has been, by the alienist touched but lightly—the analysis of the term, the definition, a passing word of comment—and then dropped. It remains, therefore that the literature of this subject is most meagre, the search for information most discouraging.

Echolalia or echophrasia, a broader and more comprehensive term, although not yet sanctioned by common usage, is a speech affection characterized by a tendency to repeat words or phrases spoken by others, hitherto most generally observed and described in combination with coprolalia³ or with palmus⁴.

The term echolalia was first employed by Romberg, who considered it an evidence of cerebral softening, but Echeverria⁵ notes it as a sign of will perversion or of impaired or defective inhibition.

Giles de la Tourette, in 1885, next takes it up and describes it in connection with coprolalia and palmus.

¹ *Exo, Echo; lalia, Speech.*

² Read before the Philadelphia Neurological Society, May 31, 1897.

³ *Kopros, Filth, the tendency to repeat foul language.*

⁴ *Palmos, A twitch, also known as Latah, Myriachit, Tic convulsif and Jumper's disease; a nervous affection characterized by localized spasmodic movements.*

⁵ Dictionary of Psychological Medicine, Vol. I., p. 424.

Noir, in 1893, made a careful study of the mental degenerates (idiots and imbeciles) of France, and asserts that the affection may occur singly as well as in combination. Landon Carter Gray⁶ coincides in this opinion, and my own experience would seem to verify the same.

Tuke⁷ gives echolalia as a symptom of the general paralysis of the insane, and adds that it may be associated with many other nervous disorders, most frequently with epilepsy—the patient in some cases imitating in his speech not only the words of the person addressing him, but the tones also.

Diligent inquiry among alienists, both here and abroad, and a careful study of imbeciles and speech defectives, covering together over eight thousand cases, gives the following data, which, while throwing but little added light upon the subject to-day, yet may by its grouping, aid in future scientific investigations.

Fletcher Beach, late of Darenth Asylum, says that echolalia was there very uncommon, although it did exist in a few cases.

Shuttleworth also found it in a few cases at the Royal Albert Asylum, but neither of the gentlemen gives statistics.

Reginald Langdon Down finds it in four per cent. of the cases of imbeciles under his care.

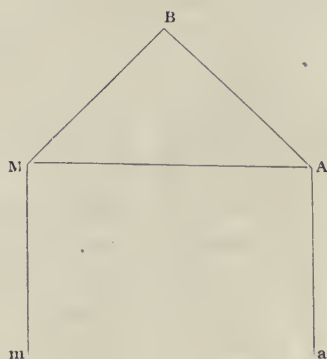
Cesare Lombroso, in a personal letter, says that he has observed echolalia in hysterical imbeciles, but never in microcephalic idiots or cretins, while W. W. Ireland's opinion in this direction narrows the field even more, as he states in a recent letter that in his experience echolalia is not found among the feeble-minded.

A case reported by Lichtheim, and by him defined as transcortical motor aphasia, may best be explained by the following diagram.⁸ Of the triangle A B M let B repre-

⁶ The American Journal of the Medical Sciences, May, 1895.

⁷ A Dictionary of Psychological Medicine, p. 526.

⁸ With thanks to Dr. Spiller.



sent the so-called concept centre (*Begriffszentrum* of the Germans), M the centre of motor images, and A the centre of auditory images. "The reflex arc consists in an afferent branch Aa, which transmits the acoustic impressions to A; and an efferent branch Mm, which conducts the impulses from M to the organs of speech, and is completed by the commissure binding together A and M." (Lichtheim, *Brain*, Vol. VII.)

"A variety of motor aphasia is created by interruption of the path B M, of which we have many examples. From the diagram we should expect the loss of

- (a) volitional speech,
- (b) volitional writing,

whilst there are preserved—

- (c) understanding of spoken language,
- (d) understanding of written language,
- (e) the faculty of copying.

So far the symptoms coincide with those of Broca's aphasia. They differ inasmuch as there is preservation of

- (f) faculty of repeating words,
- (g) writing to dictation,
- (h) reading aloud." (Lichtheim C. c.)

Charlton Bastian, referring to this says: "Lichtheim's interpretation of this case is wholly different from mine. He accounts for it by supposing a damage of commissural fibres to exist, which pass between his postulated centre

for concepts and Broca's convolution, which for him also is a motor region rather than one of sensory type."

He goes on to explain: "The meaning of this ability to read aloud in such a case is that though the auditory word-centre is so much damaged as to be unable to act spontaneously (that is, under volitional stimuli), it is still capable of responding to the associational stimuli coming to it as a result of strong excitation of the visual centre. Persons so affected are also quite capable of responding to sensory stimuli passing direct to the auditory centre itself—that is, they can at once repeat words uttered before them." (The Lancet, April 10th, 1897, p. 1016.)

(l. c.) "In this relation it may be mentioned that it sometimes happens that the speech of patients is entirely limited to a mere imitative repetition of words spoken in their hearing, while they are without the power of volunteering any statement; that is, their auditory word-centres respond only to direct sensory incitations, and not at all to those of an associational or volitional order. In these cases (usually included under the term "echolalia") a marked general impairment almost invariably co-exists.

"A defect of this kind (occurring in a woman who was hemiplegic from cerebral hemorrhage) has been recorded by Professor Béhier. She was born in Italy, and had resided both in Spain and France. Of the three languages she had thus acquired she had completely forgotten the Italian and Spanish, and had only retained a most limited use of the French. In this latter language *she only repeated like an echo* the words pronounced in her presence, without, however, attaching any meaning to them. But in the case of a woman seen at the Salpêtrière, by Bateman, the mimetic tendency was much stronger. She even reproduced foreign words with which she had never been familiar. It is clear that in such a case as this there must have been a mental degradation of a much wider kind than that which occurs when the auditory word-centre alone is reduced to its lowest grade of functional activity."

Transcortical motor aphasia, according to Déjerine, is entirely hypothetical, being in his opinion only a stage of amelioration in the cortical motor aphasia of Broca.

Mills,⁹ in 1891, cites two cases occurring in his own practice—one a woman of cultivation and refinement who would burst out with a thrice-repeated oath accompanied with an abrupt action; the other, a boy who would give unprovoked utterance to filthy language, accompanied with violent movements of the head, shoulders and arms.

Here is undoubted association with both coprolalia and palmus, as is an analogous case coming under my own observation of a beautiful and refined young girl attending a mixed boarding school, who would at intervals give sudden expression to three words successively: The first vulgar, the second foolish, the third profane; these also associated with like convulsive movements.

Again,¹⁰ Mills thinks echolalia might as properly be classed under morbid impulses as under aphasia, and describes it as "an affection in which convulsive movements are associated with sudden explosion of speech. The patient with a grimace, contortion or violent movement of some kind, suddenly bursts into an obscene, profane or absurd expression. This expression may be the echo of something overheard—hence the name, echolalia—or it may be a spontaneous outcry. It is not simply an hysterical affection, controllable and curable, but it is a true monomania, the affection of speech being beyond the patient's volition." In a still later article he classes true echolalia as a characteristic symptom of transcortical or suprapictorial sensory aphasia.¹¹

In my own personal examination of fifteen hundred and twenty-five mentally defective children, I can find but

⁹ Aphasia. Reprint from the *Review of Insanity and Nervous Disease* for September and December, 1891, p. 75.

¹⁰ *American Text Book of Diseases of Children; Speech Defects and Anomalies*, p. 663.

¹¹ *A Text Book on Nervous Diseases*. Dercum, p. 440.

two cases of what might be called true echolalia. One is not available, but the other, which is unique, I here present.

Kirtie M. Mansfield, idio-imbecile, white, male, epileptic, aged twenty-two years, with the intelligence of a child of five. He is the eldest of three children, the brother and sister being strong and healthy, both mentally and physically. Family history good, with no trace of nervous or mental disease. The parents, people of exceptional refinement and intelligence, are distantly related—the maternal mother and paternal grandmother being cousins german. Paternal grandfather died of some kidney trouble (form unknown) aged forty; maternal grandmother of some heart disease (form also unknown) aged sixty-seven. Father thirty-two and mother twenty at time of this child's birth. Born at full term, ordinary labor, nursed by mother, with no peculiarities beyond an unusually large head; a perfectly healthy infant up to sixteen months, showing, the father says, no indication of mental disease. During teething he had *petit mal*, gradually followed by prolonged spasms, and at the age of four developed true epilepsy, any excitement precipitating an attack. He began to talk with the ease of a normal child, but early developed a habit of peculiar repetition, learned the alphabet and to repeat with facility Mother Goose rhymes (which he craved to have sung to him daily). His precocious memory just at this period, coupled with these abnormal repetitions, first attracted the attention of those about him as evidencing something wrong.

In disposition he was gentle, easily governed, social, liking the presence of other children, although not joining in their plays, spending hours apart, amusing himself with blocks or weaving strings.

He had the usual diseases of childhood. In 1884 a severe attack of diphtheria was followed by vaso-motor paralysis of the left side of the face, which gradually yielded to treatment. In 1882, when he first came under the

care of the Pennsylvania Training School, he cried a great deal and talked constantly about "a nice packer o' pins and a buggy and wagon." Sight and hearing good, speech limited and enunciation slightly defective. Nervous, restless and self-willed, working himself into a fury when thwarted, muttering incoherently to himself, he spent a great deal of time twirling and untwirling a string until at last his nervous fingers found employment in knitting; in this he accomplished quite difficult patterns without assistance, himself setting up the required number of stitches, and adding as directed.

He can now count to fifty; is fond of music; is unable to read and write, but household service has proved a means of development for him, as he has learned to wash dishes, sweep and dust, and is orderly and methodical to a degree quite remarkable for one of his intellectual grade; thus he will voluntarily gather up all the litter from the floor, winding the strings into a ball, and never omits on leaving the school-room, to say: "Kirtie come to school this afternoon?" "Kirtie come to school to-morrow?" "Kirtie come to school Monday morning?" as the period may be, without once misplacing time or event. This he does day after day, invariably speaking of himself in the third person.

From this it will be seen that he has a certain amount of intelligence, although he still passes much time in a corner smiling and muttering vacant repetitions. Repeating whatever he hears, his thoughts are those of others and his speech automatic. When addressed he rarely fails in repetition before reply. Thus one may ask: "How old are you, Kirtie?" and he will immediately repeat, taking words and tones, "How old are you, Kirtie?" But here may be noted a departure from the habit of precision before mentioned. He is now twenty-two years of age, and yet to the question, "How old are you, Kirtie?" following the invariable repetition, "How old are you, Kirtie?" comes the answer, "Twelve." Though accepting the suggestion

that he is now twenty-two, he will, after a few moments, give the same reply, "Twelve." This is the only indication he gives of any loss of memory, but, indeed, I think it may rather show the presence of some strong overlaying association with that number. His keen sense of association is further shown in the following instance:

A companion of whom he was very fond, died, and, after attending a service of song some four years after, on being questioned as to where he had been, replied, "Heaven, heaven—home, Joe Zun—die song—heaven," the hymn, "Heaven is my Home," evidently recalling his loss.

His memory is, indeed, phenomenal. He recalls not only the visits of his parents and other incidents occurring during the year, but also the names of boys and attendants he has neither seen nor heard of for years, and he will sit talking to himself of them. He catches readily both words and music of all the popular songs at first hearing, repeating the words almost verbatim, or if substituting, giving equivalents.

One of the most interesting experiments with him appears all the more wonderful when we consider his low mentality. As before stated, he not only repeats words, but also imitates voice and tone of the speaker and frequently follows accurately in pantomime every movement. One afternoon I gave him, in rapid succession, words and sentences in nine different languages: English, French, German, Spanish, Italian, Japanese, Latin, Greek and Norwegian, and each time I found that, although the words were unfamiliar and would have been difficult for an ordinary person, certainly for a normal child, Kirtie took the pronunciation with facility, his voice keeping pace with mine as I repeated:

"I am here with thee and thy goats, as the most capricious poet, honest Ovid, was among the Goths."

"Liberty! Freedom! Tyranny is dead! Run hence,

proclaim it—cry about the streets, liberty, freedom and enfranchisement!”

“ Pas à pas on va bien loin.”

“ Wir seufzen im nächtlichen Winde. Vom Zweige ein Wink so fern.”

“ Superabundantissime.”

“ Vedi! le fosche notturne spoglie, de’cieli sveste l’immensa volta.”

“ Namu miò hô ren gé Riô.”

“ Potentissimus est qui se habet in potestate.”

“ Zöe mou sas agapo.”

“ Min norske vinter er så vakker, med hoida snebedakte bakker og grønne gran med pudret haar.”

On another occasion he followed me in the same words through three different tones and inflections of voice—the first a mere whisper, the last amounting to a shout, his voice always keeping tally with mine. “How do you do, Kirtie?” “How do you do, Kirtie? Pretty well.” I repeated the question in the same voice, then suddenly changing I asked the question in a loud voice: “Are you well, Kirtie?” He, expecting the other question, shouted back, “How do you do, Kirtie? Pretty well.” Realizing that his answer was automatic, and that there was no reasoning in it, I repeated it three times before he grasped the change, when he replied, “Are you well, Kirtie? Yes.” Placing my hat on the floor, I said, “Go get my hat, Kirtie.” This he repeated three times without attempting to move from his seat, seeming not to understand. Finally, picking it up and tossing it from me, I repeated the request, and, as if aroused by the action, he brought it, still repeating, “Go get my hat, Kirtie.” “Thank you,” I said. “Thank you, thank you, thank you; you are welcome.” he replied. “What did you take out of Miss Annie’s room?” “What did you take out of Miss Annie’s room? Pins. Must not steal pins to put in coat.” “What did B. B. do on the base-ball field?” “What did B. B. do on the base-

ball field? Ran away home. Bad boy," and so on, with indefinite repetition.

He is extravagantly fond of blocks, with which he will amuse himself for hours.

Some years ago he contracted the habit, when irritated, of deliberately tearing his clothing, especially his stockings, to pieces. The deprivation of his favorite plaything was found to be the best discipline for this offence. Now, when his nurse attempts to put away his blocks, he will say, "Do not take away blocks; will not tear any more." If asked if he will loan or give a block, he will reply, always repeating the question, "No, no, I will not tear my clothes," and when asked what clothes, replies, "My stockings." Occasionally, if his play is interrupted by a spasm, the blocks will be scattered, but on regaining consciousness he immediately gathers them up, knowing exactly both the position and number.

I call attention here to the fact that this case is associated with epilepsy, but neither with coprolalia nor with palmus.

Dr. William G. Spiller who has been an interested observer of the case, and to whom I am greatly indebted for aid in the work of research and comparison, says, "In performing a necropsy in a case such as you present, I should notice especially the condition of the posterior part of the left first temporal convolution. The fact that the boy is an epileptic is a point in favor of a cortical lesion, though, of course, it is no proof. As he understands all simple commands, and obeys them, the auditory centre cannot, therefore, be destroyed, but it *may* be so damaged that it is incapable of responding to volitional stimuli, yet still be capable of responding to impulses passing to it over the tract aA. I am not able to accept the concept-centre, and would prefer to explain your case in the words used by Bastian. To me your patient presents a symptom-complex resembling that of transcortical motor aphasia."

Summing up and comparing, we find echolalia a rare

form of aphasia, betokening always a marked general mental impairment, and therefore most naturally associated with other forms of degeneration.

There being no record of an autopsy of such a case, the precise location of the lesion, if there be one, is yet to be demonstrated.

In comparing the case presented with Lichtheim's proposition, we are confronted at once by a difficulty; the boy is an idio-imbecile, and his inability to read or write closes one door of observation, but we do find in common with his table, first "loss of volitional speech," and second, "preservation of understanding of spoken language, and of faculty of repeating words."

The absence of volitional speech, notwithstanding an abnormal memory, would indicate a diseased condition of the motor region, but not destruction, as he does reply and respond to word of command.

Even the repetition of words would almost appear to be such a response, or an exaggerated form of a habit of obedience to suggestion, for it is automatic, not volitional nor reflective, such as we often see in normal persons—an effort to strengthen the sensory impression so as to apprehend before acting.

Thus in the act and in the echo he is simply a creature of suggestion. His capacity for receiving such suggestions so rapidly as to echo these instantaneously without thought would tend to show less impairment of the sensory than of motor centres, and therefore confirms my impression that the defect, not so much sensory as motor, is rather to be defined as transcortical motor aphasia.

Society Reports.

AMERICAN NEUROLOGICAL ASSOCIATION.

*Twenty-third Annual Meeting, held at St. John's Parish Hall,
Washington, D.C., May 4th, 5th and 6th, 1897.*

The President, Dr. M. A. Starr, in the chair.

EPILEPSY FOLLOWING INFANTILE CEREBRAL PALSY. IMPROVEMENT AFTER CRANIOTOMY AND EVACU- ATION OF A SUBCORTICAL CYST.

Dr. Wm. M. Leszynsky, of New York, reported a case of a boy fourteen years old. At the age of two years, without known cause, general convulsions and unconsciousness occurred, being rapidly followed by left hemiplegia which still exists. Epilepsy was first manifested in his twelfth year. In the beginning the attacks were limited to conjugate deviation of the eyes and head toward the paralyzed side, with momentary loss of consciousness. These attacks increased in frequency. Later, the seizures were accompanied by rigidity of the left arm, then of the arm and leg, and ultimately there were general tonic convulsions and increasing mental enfeeblement. There was no clonic spasm at any time while under observation. After nearly a year of ineffectual hygienic and medicinal treatment an exploratory operation was advised. An opening was made in the skull over the right Rolandic area, and a large subcortical cyst was found in this region. This was completely evacuated after free incision through the extremely atrophied cortex. The brain tissue was sutured with catgut, the wound closed in the usual manner, and the patient made a prompt and uneventful recovery from the operation.

This was followed by absence of epileptic attacks for three and a half months. He then had seven general con-

vulsions, tonic in character, within eighteen hours. The cyst, which had refilled, was emptied by aspiration, and the attacks disappeared for more than three months. Again the cyst was evacuated. Bromide was subsequently administered, and under its continued use there have been only occasional mild attacks. The boy's general and mental condition have undergone considerable improvement. The speaker referred to the advisability of permanent drainage in such cases.

Dr. J. W. Putnam:—In reference to the question of convulsions being caused by pressure from a cyst, I can say that three or four years ago I had a patient, a boy between two and three years old, in whom the first convulsion was caused by trauma of the head from a fall. Some time afterwards convulsions developed. There was a second trauma from a fall in one of these convulsions. Operation was performed first over the point of injury, where he had fallen forward and struck his forehead, and a large amount of fluid was evacuated from the cyst. The fluid soaked through the thick dressing for several days. It was impossible to keep a record of the number of attacks of convulsions that the child had. They numbered fifteen to twenty in an hour. It was said that he had sometimes as many as sixty or eighty in the course of the day. That boy had a continuous loss of fluid for a week. Then the wound healed and there was no more discharge, and the patient had no convulsions for a period of several weeks, after which time they returned with the same violence and frequency. Dr. Parmenter, who made the first operation, also made the second, and we again evacuated the fluid and put in catgut and allowed drainage to go on as long as the fluid continued to flow. The convulsions commenced after a time as the flowing of the fluid ceased, and a third time the opening of the old wound was necessary. The child was last heard from six months ago and had had no convulsions whatever for two years.

Dr. Angell:—Three years ago I reported a case of imbecility and epilepsy in a child on whom craniotomy had been performed. I have recently heard from that child and desire to record the result, which has been successful after an interval of three years. Since the operation he has not had an attack of epilepsy, and there has been a steady, though rather slow, improvement in his mental condition. His habits are no longer vicious and he is gradually acquiring some knowledge and is able to take proper care of himself, and in all ways he is in a condition of satisfactory development. The pathologic condition was a hemorrhagic meningeal cyst, which offers one of the most promising opportunities for surgical interference.

In the case of subcortical cyst reported by Dr. Leszynsky, I should think that he would have to remove a portion of the cyst wall if he wished to obtain relief from the attacks.

Dr. Pershing:—Six years ago I reported the case of a man who had had a blow on the head, and some time afterwards began to have attacks of Jacksonian epilepsy. A cyst about an inch and a quarter deep was found under the upper face centre, and was drained. At the time the case was reported here only about three months had elapsed since the operation. I heard from the patient for a year after the operation, and he had no return of the attacks. After that I lost sight of him.

Dr. Worcester:—I have had one case of epilepsy, which I considered to be due to a brain cyst, in a negro, who had no paralysis at all. I found at the necropsy a cyst containing, I should think, three drachms of fluid and situated on the surface of the left frontal lobe. This seems to me to have probably been the cause of his convulsions, although there was nothing localizing in his movements.

I have had the opportunity of making seven autopsies in cases of infantile cerebral palsies, and as autopsies in these cases are not very frequently reported, perhaps it would be proper for me to speak of what I found, because the findings were very uniform. In all but one there was very great asymmetry of the hemispheres, and I am inclined to think that I may have overlooked some asymmetry in the first one, as I was not expecting to find it. There was great asymmetry of the optic thalami in that case, and the cornu ammonis of the left side was sclerotic. In four of the others the asymmetry was mainly observed in the external surface; that is, the affected hemisphere was very much smaller than the other, and the optic thalamus, and to a less extent the corpus striatum, were atrophied. In the other two cases the ventricles were very greatly dilated. In one, the brain, when exposed, showed but little difference in the two sides, but the greater part of one hemisphere was not more than one-fourth to one-half an inch thick, and the corpus callosum was reduced to a transparent membrane. In the other case the dilatation was not so great in the anterior cornu of the ventricle, but the posterior portion of the ventricle was greatly dilated, and the wall of the hemisphere greatly thinned in one place, where the external wall of the ventricle was reduced to a translucent membrane.

I judge that this must be a very common condition in such cases, and of course in cases of that sort I do not suppose that any benefit could be anticipated from operation. I speak of these cases especially with reference to the matter of prognosis in regard to operations.

The President:—I have not drained a cyst permanently: I have seen cysts drained as long as three months, and that is the

longest. My experience with cysts is an unfortunate one. I have seen the symptoms return so many times after the removal of the fluid from the cyst that I think it is useless to open them. The only thing that is of service, is to keep the cyst open so that it fills up from the bottom. In the long run operations on cysts of the brain, whether of traumatic or unknown origin, are almost futile. I base this statement on my experience in eight cases; two of them, which have been operated on within three months in spite of my semi-approval of the operation, have shown that it is practically useless, and in none has there been permanent recovery from the epilepsy.

Dr. Wm. M. Leszynsky, of New York:—We all know that, as a rule, these are the most unfortunate cases for surgical treatment. However, in a case of this character, I think we are justified in recommending an exploratory operation.

In the case reported, considerable relief followed each evacuation of the cyst, and the improvement has been permanent.

Dr. Graeme M. Hammond read by title a

REPORT OF A CASE OF JACKSONIAN EPILEPSY, RELIEVED BY AN OPERATION.

Dr. Chas. L. Dana, of New York, read by title the following paper:

TWO CASES OF BASEDOW'S DISEASE, WITH AUTOPSIES.

Dr. Samuel Ayres, of Pittsburg, read a paper entitled:

AN UNCOMMON NASAL PARESTHETIC NEUROSIS.

Dr. Baker:—A patient of mine, a woman approaching the menopause, has a similar sensation in the tongue, recurring attacks of fullness and pain, and disagreeable sensations in the tongue, that come and go. Sometimes they will be absent for a number of weeks, then, again, they will recur frequently for a few days, and then will be followed by a longer period of decline and a final subsidence, and so on.

I have always found in the management of her affection that sharp faradization of the tongue stops the attack. She is of a neurasthenic constitution and degenerate type. I have no

theory to advance. The thought occurred to me that possibly the condition of the nose in the patient of Dr. Ayres may be allied to that condition of flushing of the eyes and smarting of the lids which is found connected with the rheumatic or gouty diathesis. If I had such a case I should attempt to treat it with this idea in mind.

Dr. E. W. Taylor, of Boston, read by title a paper on

FIVE DEFECTIVE BRAINS.

These brains were all taken from persons who showed more or less unmistakable signs of insufficient mental development. The exact determination of mental deficiency was, in certain of the cases, difficult, owing to the extreme youth of the patients. The brains show marked alteration in gross structure, to which fact we desire now to call special attention, rather than to the more fundamental microscopical alterations, which it is hoped may form part of a more complete piece of work to follow this preliminary report.

Case I.—The brain is from a young child from the Infants' Hospital, who was supposed to have suffered a fracture of the skull in the frontal region. Operation was performed and was followed by death. The brain presents a remarkably symmetrical, bilateral agenesis of the frontal lobes, of the type of microgyria. The remainder of the brain, macroscopically, is essentially normal. The sharp line of demarcation between the atrophied and the normal convolutions, suggesting an intra-uterine affection of the anterior, and part of the middle cerebral arteries, as the cause of the maldevelopment, is of particular interest. The resemblance of this brain to that of the higher apes is exceedingly striking.

Case II.—This brain is from a microcephalic idiot of twenty years, who had neither spoken nor walked during life. The specimen shows a defective development of both hemispheres, but chiefly of the left. Microgyri were marked in the occipital region. The fissure of Rolando and the general arrangement of the convolutions are entirely anomalous. Of chief interest is the extreme thinning of the wall of the hemispheres in both parieto-occipital regions, again most markedly in the left, with a con-

sequent dilatation of the posterior horns of the lateral ventricles. The brain does not cover the cerebellum, which is well developed.

Case III.—The brain is from an infant of approximately eight months, from the Infants' Hospital. Clinically, the child showed signs of defective mental development, which was associated with frequent convulsive seizures of a peculiar character.

The necropsy showed a brain practically non-convoluted, a condition which we believe to be of most extreme rarity. There are no marked gross defects in this case; the cerebellum is well covered by the hemispheres, and the island of Reil is not exposed. Apart from the fissure of Sylvius, which naturally must be present, and the first temporal sulcus, there are no fissures nor sulci whatever on the convexities of the brain, except a few shallow and anomalous furrows. The fissure of Rolando is absolutely lacking. The brain has the general appearance of being covered with plaster. The mesial aspect of the hemispheres is slightly fissured, as is also the base. The cerebellum and brain stem are normal in appearance.

Case IV.—The brain is from a child of two years, from the Infants' Hospital. There had been marked mental defects. Operation of craniotomy was performed and repeated, but death occurred after the second operation.

The specimen is also one of great rarity, though not so unusual as the one preceding. There is complete absence of both cerebral hemispheres, with the exception of imperfect occipital lobes, and a small portion of the temporal lobes. The central ganglia are present; the optic thalami are poorly developed; the cerebellum is well formed and of normal size; the pons is small, and agenesis of the pyramidal tracts is observed in the medulla oblongata.

Case V.—The brain is from a microcephalic child of three months, from the Infants' Hospital. In this case a small tumor, apparently connected with the dura mater, projected from the vertex of the skull. Operation for its removal resulted in death. The necropsy showed a small brain, chiefly anomalous in the extreme lack of development of the whole right cerebral and cerebellar hemispheres, with a consequent distortion of the left hemisphere. The longitudinal fissure of the brain describes a

curved course, its concavity is toward the atrophied hemisphere. The volume of the right hemisphere is approximately a third of that of the left. The same is true of the cerebellum. The convolutions are entirely anomalous in both hemispheres.

Dr. Wm. C. Krauss presented a paper with the title
A RECEPTACLE FOR HARDENING HUMAN BRAINS.

Dr. Charles W. Burr read by title

A CASE OF PSYCHIC ANESTHESIA.

B. C. was twenty-four years old when he presented himself to Dr. Burr for treatment. When he was about ten years old he was accidentally struck on the side of the head by an axe handle with such force that he was thrown into a river, on the bank of which he had been standing. Examination of the head showed that he had a simple depressed fracture of the right parietal bone over the motor area. He remained in a state of alternate coma and delirium for about three weeks. On recovering consciousness he found himself partially paralyzed on the left side of the body and face, and completely anesthetic upon the same side. The palsy and anesthesia entirely passed away in a few months, sensation returning before motion. He was supposed to have recovered completely, until, on putting his left hand into his coat pocket for the first time after his illness, he discovered that he could not tell what he had in his grasp, though he had preserved the sense of touch. Little attention was paid to this symptom at the time, and he was told that it would soon pass away; but it has not done so.

The present examination shows that he is a fairly healthy-looking man, though neurotic, supersensitive and morbid. The left leg, arm and face are slightly smaller than the right. There is no palsy of either side of the body, but the left hand is used a little awkwardly. The gait and station are normal. The knee-jerks are equal and a little exaggerated. There is no depression or pain on pressure at the seat of the alleged fracture. Pressure on

the vertex over an area about as large as a one-cent piece causes mental confusion, a condition of dreaminess, and, if long continued, light hypnotic sleep. With the eyes shut he recognizes well variations in the positions of the hands or arms. Tactile sense is normal on both sides, except that on the entire left side, even on the finger tips, he fails to localize touch. He is absolutely unable to recognize any object put into his left hand, but knows that he is grasping something. His grasp is good, and remains good when the eyes are shut, there being no muscular relaxation even after several minutes. There is no sensory trouble in the right hand. On both sides the temperature and pain senses are normal, and he can distinguish dull objects from sharp ones. There is no difficulty with speech, vision, hearing, taste or smell. The urine is normal. Examination of the thoracic and abdominal viscera is negative. There is a partial reversal of the color fields.

This case differs from similar ones in the loss of the ability to localize sensation, and this, the writer believes, stands in causal relation to the failure to recognize objects by touch. He regards the case as probably one of hysteria.

Dr. Smith Baker read a paper, entitled:

STEPS TOWARD INSANITY.

He stated that recent studies of the neuron seem to indicate that the biological doctrine that activity determines structure, and thus, in turn, determines function, may be applied to the causation of insanity. The rule seems to be that exhaustion of the brain cells comes first, then acute intoxication, and finally, structural changes as the result of these conditions. The major premise of every study of the causation of insanity may be assumed to be this, viz.: every pathopsychical manifestation in the individual is evidence of neuronie structural defect, and until otherwise proved, every neuronie structural defect should be regarded as evidence more or less conclusive of remote untoward influence primarily on the part of ancestry. The neuronie defect itself, according to Van Gieson, may always be regarded as a true parenchymatous degeneration, involving not only the cells proper, but primarily their

ultimate protoplasmic expansions and "contact granules" (Andriezen). With reference to the steps by which vesania is initiated, we must look to ancestry for the first ones. Marriage of unmarriageable parties results in certain tensions and stresses which lead to arrests and perversions of development on the part of children. These, not generally presenting evidences of vesania themselves, carry over to succeeding generations their own hereditary defects of structure and function, and in the latter they become intensified and eventually break out in pathopsychical manifestations. Probably one-third of all marriages are of a character which necessitates a bad prognostication as regards progeny. Again, overstrain, worry, nutritional perversions, and toxemia resulting from these during the child-bearing period, are other sources of vesanic predisposition. The same should be said of the inadequate training to which so many children are subjected. Accidents, diseases and emergencies serve as exciting causes chiefly where birth, nurture and education, singly or combined, have been deficient. All this suggests a prophylactic pedagogics founded upon neurological conclusions.

PHILADELPHIA NEUROLOGICAL SOCIETY.

October 25th, 1897.

The President, Dr. Charles W. Burr, in the chair.

Dr. A. Ferree Witmer exhibited

A CASE OF AKROMEGALY.

The patient was a female, aged fifty-five, unmarried, and a native of Ireland. The early personal and family history were negative. The time of onset of the disease was unknown. A photograph of the patient, taken in 1876, showed disproportionate development of the lower part of the face, including the pinna of the ear. The woman applied for treatment of a dull, intermittent pain in the left knee joint. The joints throughout the body were fully movable and non-crepitant. She complained also of somnolence so intense that she frequently fell asleep while at work. This condition had been noted for two years, and was as likely to occur in the morning as in the evening hours, and was increasing in degree. She had complained of vertigo for a period of six months, about two years ago, and had a dull morning headache also at that time. The tongue was swollen at times. The voice had become deeper in pitch, the speech slow and muffled, the appetite slightly in excess, and the thirst constant. The urine was voided frequently and in large quantities. She had intractable diarrhea at irregular intervals, and had had the menopause twelve years ago, and no abnormality had been noted throughout its course. She had hot and cold flushes; her extremities were always comfortably warm. She was not oversensitive to heat or cold; her memory had failed slightly; she had worn a number five boot comfortably five years ago, but a number seven was now required. There were no disturbances of the special senses.

The weight of the patient was two hundred pounds; her height was five feet six inches.

The enlargement was general and not circumscribed to the extremities. Skiographs of the hand and foot showed marked increase of the soft parts, possibly with changes in the cartilages, but with no increase of the bony structure. Fluoroscopic examination of the lower jaw and of the thorax showed proportionally no increased amount of bone. Her hair was abundant and natural in color; her skin was puffy and pitted slightly on pressure over the dorsum of the hands; the face was elongated but symmetrical; the eyelids were enlarged; the nose was thickened; the cheek bones were prominent; the cheeks were flattened; the lower lip was protruded; the tongue was large, flabby and slightly fissured; the palate was highly arched; the cartilages of the larynx and nose were hypertrophied; the lower jaw was very prominent; the neck was short and thick, and the thyroid body was not prominent. There was no retrosternal dulness; there was slight cervico-dorsal kyphosis. The measurements of the trunk and extremities gave normal averages in length, but the width was materially increased. The nails of the fingers and toes were normal in appearance; the heart and lungs were normal; the respiration was shallow; the urine was of low specific gravity, 1008 at one examination, but contained no albumin, no sugar, and no peptone; the eyeballs were slightly prominent; the visual fields for form and color were normal; the pupils responded freely to light in accommodation and in convergence, and a far-sighted astigmatism of mild degree was present. Tests to determine rate of sense perception gave high thresholds in every instance, showing an average retardation of reaction time to forty per cent. below the normal. Dynamometer tests indicated average strength. The knee jerks were sluggish; there was no clonus; the station was normal, and the gait was quick. The medication consisted of an extract of thyroid body in doses of fifteen grains daily for a period of three months, but this was without benefit—and later of eight grains of an extract of pituitary body daily for two months, which also was without apparent benefit.

Dr. Spiller exhibited, in the names of Drs. F. A. Packard and H. W. Cattell,

THE BRAIN AND SPINAL CORD FROM A CASE OF
AKROMEALY.

He stated that Strümpell had recently remarked on the great number of cases of akromegaly in which tumor of the pituitary body had been found.

The specimens presented were from a case which had been reported clinically by Dr. Packard. A large tumor which proved to be a round-cell sarcoma, and was about the size of an English walnut was found in the pituitary body, and had eaten away the base of the skull by pressure. The optic nerves were much pressed upon, and the nasal side of the right nerve was degenerated. The left had not, at that time, been examined. Large calcareous plates were found in the pia-arachnoid of the cord, and were very numerous. These were not supposed to have been in relation with the akromegaly.

The speaker stated that it is difficult to believe that a small gland, such as we know a part of the pituitary body to be, could by its altered functions produce the appearances of a systemic disease like akromegaly, though he could not deny the possibility of this. He said that occasionally the gland had been found diseased when the symptoms of akromegaly had not been present, as in a case reported by Dr. Packard in connection with this case of akromegaly. The frequency of disease of the pituitary body in cases of akromegaly, he thought, did not prove conclusively that this is the cause of the latter affection.

He stated that he had been much interested in the possibility of surgical interference in cases of akromegaly in which the symptoms rendered the diagnosis of a tumor of the pituitary body probable, as in this case, in which bilateral temporal hemianopsia, with optic atrophy, intense headache, somnolence, absence of the patellar reflex, and failure of memory, were present. In other cases in which the diagnosis of tumor could not be made, the question of operation, of course, would hardly arise. If we believe that altered function of the pituitary body is the cause of akromegaly, he thought that we might well dread the effects of removal of the entire gland. He regretted that

Dr. Keen had been unable to be present to discuss this important question, and read the following communication from him:

"You are at liberty to say that my experience in the case of brain tumor in the frontal lobe, operated on in Baltimore a year ago last spring with Dr. H. M. Thomas, and shown at the meeting of the American Neurological Association here not long afterward, would make me believe that by lifting an osteoplastic flap from the forehead, either in one large piece or possibly better by one flap, on each side, it would be possible to reach the pituitary body. Whether the hemorrhage and other emergencies that might then arise would be too difficult to cope with, I do not know, but I should believe that we could do so successfully."

DISCUSSION.

Dr. F. A. Packard:—I have nothing particular to add to what Dr. Spiller has said. The condition of the patient remained practically unaltered from 1892 until the last time I saw him, six months before his death. Between 1886 and 1896, there was no return of the attacks of somnolence. If the somnolence had been due to the tumor, is it presumable that it would have continued.

There was no loss of smell when I last tested it a year ago.

There was one symptom which was never sufficiently studied, because it was difficult to get hold of the man, and that was the profuse secretion from the nose. The patient had constant severe headache which brought tears to his eyes, and it was a question whether this secretion was not composed in great part of tears. On one occasion I found that this fluid contained a large quantity of sodium chloride, a good deal of nucleo-albumin, and under the microscope a large number of mucus corpuscles.

The fields of vision were tested a second time in 1894 or 1895 by Dr. de Schweinitz and found to be contracted in all directions, though previously there had been bitemporal hemianopsia. There was no material change from the report in 1892. I ascribed the increase in visual power to necrosis of the sella turcica allowing more room for the spreading out of the nerve fibres, although this explanation did not seem to be entirely satisfactory. It is possible that a hemorrhage occurred in the tumor in 1885, giving rise to increase of bulk of the mass, and so producing somnolence and hemianopsia which ceased as the blood was absorbed.

Dr. Francis X. Dercum:—I am glad to see these interest-

ing specimens as I saw the patient many times during life. I am also pleased to hear that Dr. Spiller takes the position that I did some years ago, namely, that it is not philosophical to ascribe the symptoms of akromegaly to disease of the pituitary body. As a matter of fact, there is scarcely a gland in the body that has not been described as enlarged in certain cases of this affection. It, therefore, seems premature to ascribe it to disease of the pituitary body, even if we exclude those cases in which this has been found to be normal.

Dr. A. A. Eshner:—I should like to say in connection with this subject that Woods Hutchison, in reporting a case of akromegaly, with enlargement of the pituitary body in a giantess (*American Journal of the Medical Sciences*, 1895, p. 190) suggests that at least one form of giantism is merely akromegaly beginning in foetal or infantile life. He offers the further suggestion that the nutrition of the body may be presided over by structures in the pituitary region. A series of measurements showed that while the pituitary fossa in the skulls of cretins and dwarfs is markedly contracted in size, it is unusually enlarged in cases of giantism and akromegaly, as well as in the skulls of the anthropoid apes, which present some features of akromegaly.

In reference to the question of surgical interference, I would call attention to the fact that in the *British Medical Journal* for 1893 (No. 1722, p. 1421) Caton reported a case of akromegaly with symptoms of intracranial pressure. An opening was made in the skull with relief of suffering and prolongation of life. Operation had been recommended earlier but was not at first consented to. It was hoped that by opening the skull the intense pain could at once be relieved, and subsequently the enlarged pituitary body be removed. Accordingly the bones forming the anterior portion of the right temporal fossa were removed, but at no time did the condition of the patient appear suitable for the more radical operation. Upon post-mortem examination the pituitary body was found to be as large as a tangerine orange and presenting histologically the structure of a round-cell sarcoma. In commenting on this report at the time, I took occasion to say that "the unfortunate outcome of the case . . . lay in the nature of the case itself, rather than in a failure of the operation or of the principles on which it was based, . . . and in future the propriety of operation in selected cases of akromegaly should receive due consideration."—(*Medical News*, Jan. 27th, 1894, p. 108.)

Dr. A. Ferree Witmer:—In the case that I present, perverted sleep has been a marked symptom, but there probably is no tumor as the eye grounds are normal, there is no staggering gait, and the knee jerks are normal.

Dr. James Hendrie Lloyd reported

A CASE OF TOTAL AND COMPLETE UNILATERAL OPHTHALMOPLEGIA (BOTH EXTERNAL AND INTERNAL).

The patient, a man, aged thirty-eight years, had at first what appeared to be simply a paralysis of the third nerve of the right eye. Later, however, the fourth and sixth nerves also became involved. The eyeball was absolutely immobile and was directed forward. There was complete ptosis, and the pupil did not respond to light or on accommodation. Severe headache was felt, and this was located above and behind the orbit. Slight exophthalmos was noticed later. The sensory tests were of great interest there was complete anesthesia of the conjunctiva and in the whole territory of the supraorbital nerve as far back as the vertex. Both the upper and lower lid were anesthetic on their edges, except towards the inner canthus, and this indicated that the nasal branch of the ophthalmic nerve was not completely involved. This was shown also by the fact that the mucous membrane on the interior of the nostril and the small patch of skin on the nose supplied by this branch were not anesthetic. There was retardation of tactile sense, but not complete loss, in the territory of the superior maxillary nerve. The inferior maxillary division of the fifth was entirely exempt, as was also its motor branch. The seventh and eighth nerves were not involved. There was a slight choking of the disk of the affected eye. The diagnosis of a syphilitic lesion just behind the orbit was made. The autopsy revealed a gunmatous inflammation behind and extending into the orbit. It had also invaded the walls of the cavernous sinus. The Gasserian ganglion was not included in the growth, and the foramen rotundum and foramen ovale, through which pass respectively the superior and inferior maxillary nerves, were just on the border of the affected area.

ABSTRACT OF APÁTHY'S VIEWS ON THE STRUCTURE OF THE NERVOUS SYSTEM.

This was the title of a paper read, on invitation, by Thos. H. Montgomery, Jr., Ph.D. (Berlin) of the Wistar Institute of Anatomy and Biology.

He said that the structure of the axis-cylinder process has been the object of much discussion, and an equally mooted point is the question as to direct connections between the processes of nerve cells. It is here intended, after a brief historical introduction, to present, without criticism, the results contained in a recent paper by Stefan Apáthy: "*Das leitende Element des Nervensystems und seine topographischen Beziehungen zu den Zellen*," which appeared in the *Mittheil. d. zool. Station Neapel*, vol. xii, 1897.

The protoplasm (cytoplasm) of the ganglion cell is acknowledged to consist of a denser and a more fluid substance; but the various observers have proposed most divergent views as to the arrangement of these two constituents. Thus Leydig concludes that the denser substance, his spongioplasm, is arranged in the form of an irregular, spongy network in the more fluid hyaloplasm. Hans and Max Schultze, with perhaps the majority of authors, consider the denser portion to be arranged in the form of fibrils. Flemming, that the denser substance, his mitom, occurs in the form of short and isolated fibrils in the fluid paramitom. Bütschli considers the structure of the cytoplasm to be alveolar, and other authors, to consist of granula. Rohde stands alone in assuming the denser substance of the ganglion cell to be produced entirely by strands of neuroglia fibres, which penetrate into the cell body to form a component part of the same.

The structure of the axis-cylinder has, likewise, been most variously described; but we will not take the space here to present more than a few of the more representative of these views. The prevalent idea, founded particularly by Max Schultze, is that it consists of a bundle of primitive nerve fibrils imbedded in a homogeneous matrix; these fibrils entering the cell body, and according to some of the observers, encircling the nucleus. Leydig holds that the hyaloplasm is "*die eigentliche Nervenmaterie*" (the real nervous material), and alone forms the core of the axis cylinder, which is enveloped by a sheath of spongioplasm (not to be confused with the sheath of Schwann, formed by external neuroglia fibres). In a recent study on the elements of the central nervous system of the *Nemertini* (a group of worms), I reached essentially the same conclusions; and indeed this view has been adopted by many investigators of the invertebrates. Nansen

in a brilliant paper modifies the view of Leydig, describing the axis cylinder as consisting of a bundle of nerve primitive tubules, each such tubule formed of a hyaloplasmic core and a spongioplasmic sheath. Bütschli holds that the primitive fibrils are nothing but much elongated rows of alveoles.

As to anastomoses or direct connection between the processes of ganglion cells, it may be said to be the most generally accepted modern view that such do not exist.

Apáthy has studied principally the nervous system of the leech (*Hirudo*) and of the earth worm (*Lumbricus*); but reached essentially the same conclusions for these cells in other *Hirudinea* and *Mollusca*, and in vertebrates, in *Lophius*, *Triton*, the rabbit, and the ox. He maintains that his own methods of preparation, which are the results of years of careful experimentation, are the only ones adequate for the clear differentiation of his neurofibrils. These are (1) a gold impregnation method (after fixation); (2) a methylene blue staining method, and (3) staining with a certain hematein solution. These methods are explained fully in his recent paper (with the exception of the previously-described methylene blue method), and though the *modus operandi* of each is complex, and necessitates especial adaptation for each object to be studied, it furnishes, nevertheless, very clear and beautiful preparations.

Apáthy distinguishes between nerve and ganglion cell as follows: The nerve cell is the producer of the neurofibrils, and hence the producer of that which conducts; while the ganglion cell produces that (force) which is to be conducted; "a division of labor, which has differentiated the neuroganglion cells, with both kinds of functions, into these two cell forms which differ histologically and histogenetically."

According to him there are various kinds of connections between ganglion cells serving to place them in direct and indirect conjunction, and such anatomical connections are histogenetically referable to intercellular bridges. (1) Two cells send a process (either dendritic or axonic) into the same nerve; or (2) the processes of several cells join to form one, or one cell is apposed to another; or (3) a process of one cell is joined with the cell process of another, or a number of such intercellular bridges are pres-

ent, or (4) two cells are united by their collateral or terminal branches; or lastly (5), and this is "the principal mode and means of the conducting connection," the processes (axis cylinders) of two cells pass over, by continued ramification, into an anastomosing lattice-work. All these types of ganglion cell unions were demonstrated by his preparations.

He finds that the neurofibrils are produced by the nerve cells, and in the leech also by the neuroglia cells, so that in this animal, at least, neuroglia cells must be regarded as nervous and not as simple connective tissue elements. Such neuroglia cells may produce both neurofibrils and neuroglia fibrils, or only one of these kinds of fibrils. These two kinds of fibrils are easily distinguished from one another and from connective tissue fibrils by his staining methods. Each neurofibril consists proximally of a bundle of elementary fibrils; but in the course of ramification of the former, bundles of these elementary fibrils are given off, so that at the distal end of a neurofibril only a single elementary fibril remains. The course of the neurofibrils is undulatory. Varicosities of nerve fibres are artefacts.

Now the most important result of Apáthy's studies is that a neurofibril, arising in some particular nerve cell, passes out of one of the processes of the latter, and in its further course may transverse several ganglion cells, and passing out of these again, finally terminate in or around a muscle or sense cell. In the leech the ganglion cells are small and close together, and these results were derived from a study of thick sections, in which only the neurofibrils were deeply stained. From the importance of this conclusion it is necessary to consider more in detail the mode of distribution of neurofibrils in ganglion cells.

In the leech (*Hirudo*), the object most fully studied, he found the body of the ganglion cell to consist of the following layers, enumerating from the outside: (1) the outer glia zone; (2) the inner glia zone; (3) the outer alveolar zone; (4) the outer so-called "chromatin" zone, the granules of which are chemically comparable to chromophilic granules; (5) the inner alveolar zone; (6) the inner "chromatin" zone, connected with the outer corresponding zone by radial bridges; and (7) the perinuclear zone, in which is a corpuscle comparable to a centrosome. The

"*Stammfortsatz*" of the cell does not correspond alone to the axis-cylinder process of the vertebrate ganglion cell, but to axis-cylinder, plus dendritic processes (these cells are unipolar); this process consists, apart from the contained neurofibrils, of a dense, almost homogeneous substance.

According to the mode of distribution of neurofibrils in ganglion cells, Apáthy distinguishes two types of cells: (G) that of the larger, and (K) that of the smaller cells. In the first type the neurofibrils form a lattice work within the outer "chromatin" zone of the cell, and are irregularly grouped in the whole diameter of the axis cylinder; they come out of this process, coursing mainly meridionally, and after continued ramifications and anastomoses reach the apex of the cell, and then, on the opposite surface of the "chromatin" zone of the latter, collecting together again, pass out into the cell process. "The cell process contains accordingly cellulipetal as well as cellulifugal neurofibrils, and in the cell body the cellulipetal neurofibril passes directly over into the cellulifugal." This first type of cells contains no inner "chromatin" zone. In the second type of cells the neurofibrils are grouped into two zones, concentric hollow spheres of anastomosing fibrils, these two zones being connected by radial neurofibrils; (1) the perinuclear lattice work, which lies in the cell body at the boundary of the perinuclear and inner alveolar zone; and (2) the perisomal lattice work, which lies in the outer "chromatin" zone of the cell.

In this second type of cells, then, the cellulipetal fibrils pass from the cell process into the perisomal lattice work, composing the latter, and from there pass radially to the inner, perinuclear lattice work, and at the distal pole of the latter converge to form a single, thick, cellulifugal neurofibril. "The thick, axial primitive fibril of the cell process accordingly represents the cellulifugal, and the more peripheral, thinner primitive fibrils the cellulipetal portion of the conduit, and the whole ganglion cell of type K is most probably motor." That is, the thicker neurofibrils are motor, the thinner, sensory. "No primitive fibril passes through the ganglion cell without having ramified and taken part in the production of a lattice work within the cell body. And, on the other hand, no entering primitive fibril terminates in any way within the cell body.

nor does a departing fibril arise in the ganglion cell." Essentially the same conclusions were reached also for the ganglion cell of *Lumbricus*, and cells of the spinal chord and medulla oblongata of *Lophius*, *Triton* and *Bos*.

In *Lumbricus* the large cells of Leydig are not ganglion cells, though they may be nerve cells.

The terminal neurofibrils, on reaching nerve and muscle cells, enter these cells but do not terminate within them, but leave them again to anastomose around them. In ciliated epithelia the neurofibrils appear not to enter the cells, but to form an intercellular lattice work.

Such are, as briefly as possible, the most striking results of Apáthy's researches. His figures seem to be quite convincing, and more than one colleague, on seeing his preparations, has been won over to his views. The main object of this review has been to make American neurologists acquainted with this important paper, which might not, otherwise, have become so quickly known to them, since it was published in a zoological journal. We should not remain skeptical as to the truth of his results, but rather test their validity by using the microscopic methods recommended by him. For if these results be corroborated, the structure of the nervous system will be placed in an entirely new light, necessitating a complete reversal of most of our present ideas.

DISCUSSION.

Dr. Chas. K. Mills:—This is one of the most important communications ever made to this Society, and while I do not feel competent to discuss the matter from the standpoint presented, I cannot fail to express my thanks to the speaker for its presentation. As has been said, these views are not only reactionary, but revolutionary, and if confirmed, will compel us to recede from the views recently adopted as to the nerve cell as an anatomical unit. Our theories of the neuron must disappear or be largely modified. We must wait for a full confirmation of views so revolutionary.

Dr. Spiller:—Possibly many here present will share the incredulity I experienced when I first heard these statements. When I learned, however, that Apáthy had been working on these investigations many years; when I read a letter from one of the most famous histologists in the world confirming his statements; when I heard that his specimens had been examined by many men and had been acknowledged by them

to be demonstrative; when I saw the careful manner in which the paper had been prepared; it seemed to me that the subject was too important to be dismissed lightly.

If these views are accepted they will upset our present conceptions. Anastomoses of nerve cells! We are going back to something resembling the old views of Gerlach. It is not easy to believe that neuroglia cells are nerve cells. We know that neuroglia cells and nerve cells have embryologically a common origin, but we have believed that in their full development they are very different structures. Fibrils within the protoplasm of the ganglion cells (in the old sense) have been described by many. A friendly strife has arisen between Lenhossék and Flemming. The former at one time stated that he was unable to find the fibrillary structure in the axis cylinder of the spinal ganglion cell, but this year he has published a careful study on the spinal ganglion cells of man in which he acknowledges the existence of fibrils within the axis cylinder. He has been unable to observe them within the cell body. Flemming has replied to this paper and has stated positively that they are present also within the latter.

Van Gehuchten, at the recent congress in Moscow, read an important paper on the structure of the nerve cell. He says that ganglion cells contain a network on which there is an incrustation of chromophilic substance; that in certain places this incrustation is sufficient to produce the Nissl corpuscles; and that the vacuoles, which have been observed within these corpuscles, are the meshes of the network which have not been filled by chromophilic matter. I do not find, however, any reference to Apáthy neurofibrils in this report.

Some years ago Marinesco advanced the theory that a nerve cell (in the old sense) is only kept in a normal condition by the reception of impulses from the periphery of the body and from the brain. If either source is cut off, the cell suffers. A short time ago I had occasion to express the opinion that the possibility of tertiary degeneration in cerebral hemiplegia had never been irrefutably demonstrated. Soon after I had made this statement, Schaffer's paper, in which changes are described which occurred in the motor cells of the spinal cord in hemiplegia forty-eight days after the beginning of the attack, came into my hands. He believes that the atrophy of hemiplegia is the result of destruction of motor fibres in the pyramidal tract. These views come to us in a new light if we may believe that there is direct continuity of neurons by means of neurofibrils, and not merely a loss of impulse to motor spinal cells.

As another illustration; Marinesco described changes in the cells of the posterior nucleus of the vagus from peripheral

lesions of this nerve and concluded, therefore, that this nucleus must be motor. Van Gehuchten states that he has found changes in the acoustic nucleus of the oblongata a short time after section of the eighth nerve. The axis cylinders which pass into this nucleus, *terminate* about its cells. He believes that changes occur in the cell body of the central neuron after lesion of the peripheral neuron. If we dare believe that neurofibrils pass from the first neuron into the cell body of the second, we can readily understand why there should be an alteration of structure in the latter.

Let us look at the subject of the reflexes in the light of Apáthy's discoveries. If it is true that neurofibrils pass from sensory fibres into motor cells, we may be able to better understand the difficult subject of the reflexes. It is not unusual in great exaggeration of the reflexes to obtain contraction of the adductor muscles of one thigh by striking the patellar tendon on the opposite limb. A reflex is not limited to one or even a few muscles. If neurofibrils pass through a number of ganglion cells, we may readily understand how an impulse acts on many cells.

Dare we believe that the many neurofibrils which leave a sensory cell, in contrast to the single one which leaves a motor cell, are indicative of the conveyance of different forms of sensation?

These are only a few thoughts which occur to me. I am seeking light, not stating facts, and am well aware of the danger of theorizing, but time will demonstrate the truth or fallacy of these suggestions.

NEW AESTHESIOMETER.

In the June number of the Bulletin of the Johns Hopkins Hospital, Dr. L. F. Barker describes an ingenious little instrument recently prepared by Professor Von Frey for studying pain and pressure sense. It is an improvement on an apparatus previously described from Von Frey's clinic by Dr. Barker, in which the amount of pressure necessary to bend test hairs of various diameters was employed. The new instrument has the advantage that with a single hair one can obtain a number of different pressure values.

It consists of a long hair pushed through a capillary tube like a thermometer tube; by sliding a sheathing over the tube it may be made to project more or less. According to the length of the projection of the hair is the strength which is necessary to make it bend, and a reading of the scales inscribed upon the tube gives thus some idea of the force used. Dr. Barker quotes a case in evidence of its usefulness, in which ordinary slight stimuli appeared to cause pain. It was supposed that pressure sense was absent. It was easy with this instrument to prove that pressure sense was not abolished, though the level for pain was almost the same as that for touch. The importance for such an observation is obvious for showing the retention of tactile sense in cases where it is obscured by hyperesthesia. MITCHELL.

NEW YORK NEUROLOGICAL SOCIETY.

Stated Meeting, November 2d, 1897.

Dr. C. A. Herter, the Vice-president, in the chair.

THOMSEN'S DISEASE.

Dr. George W. Jacoby exhibited a typical case of Thomsen's disease. The patient, a young man, had been referred to him by Dr. Schwinn, of West Virginia, with a correct diagnosis. The patient was twenty-eight years of age and had lived in this country since 1884. There was nothing in the family history bearing upon the condition especially, except that a distant cousin was said to have walked stiffly and in a peculiar manner for fifteen years. The patient himself had always been delicate, but had been as active as other boys. He had had typhoid fever in 1889, and on recovering from this had first noticed a cramp in the legs. After a little it was found that he could not execute movements as quickly as before. In 1893, he first sought treatment. For the past year or two his arms and hands had also been affected. The condition varied considerably at different times, but was apparently not affected by meteorological changes. The examination showed quick reaction of the eye muscles, with spasm of the external rectus; cramp of the masseter muscles on bringing the jaws together forcibly; no involvement of the pterygoids. All the muscles of the upper extremity and of the thorax were involved—indeed, nearly all the muscles of the body. The contraction of the muscles was decidedly tetanic, and was very marked at first, but, on repeated tests, it gradually subsided. The electrical examination showed marked myotonic reaction, and also a wave-like appearance, but it was not certain that this latter phenomenon consisted of a series of waves, such as are observed in water. A piece of muscle had been excised from the biceps, and also from the quadriceps, but they have not yet been minutely examined. The case was quite characteristic on account of the marked variations

occurring from time to time. The speaker said that in an article, published by him ten or more years ago, he had taken the stand that these cases were probably of myopathic origin, due to some congenital defect in development, but in the light of modern investigation he was now disposed to believe that some central cause was at work—that there was a functional hereditary derangement of the central nervous system—a condition of lessened resistance in the cells. This did not seem to him a strange assumption, when one considered the well-known idiosyncrasies exhibited to various toxic influences. On the theory that some kind of toxæmia was at the foundation of this disease, he thought the observed phenomena could be explained—at least in this direction seemed to lie the possibility of solving the pathogeny of this class of cases. This patient had not been affected by the disease until eighteen years of age; hence, there was no propriety in calling such a case “*myotonia congenita*.” He would divide these cases into three classes, viz.: (1) *Myotonia congenita*; (2) *myotonia acquisita*; and (3) *myotonia transitoria*.

DISCUSSION.

Dr. Frederick Peterson asked why a theory of causation might not be founded upon chemical changes in the muscles? Changes in the structure of the muscles, he said, were known to arise—for instance, in connection with typhoid fever.

Dr. Herter thought that we must look to toxic agents as furnishing at least a clew to the causation of such conditions. The peculiar susceptibility to certain types of poisons, seen, for, instance, in epilepsy, must be referred to peculiarities of the central nervous system. He would agree with Dr. Peterson that these cases did not seem to be of central origin, and that it was more probable that they arose from chemical changes in the muscles. To study this subject successfully, it would be necessary to inquire into the condition of the secretions and excretions at the time of the onset of the disease, and not after it had become chronic.

PACHIMENINGITIS HEMORRHAGICA INTERNA IN CHILDREN.

Dr. C. A. Herter said that internal hemorrhagic pachymeningitis was usually considered to be a very rare condition in children, yet one German observer had found it

in about 17 per cent. of his autopsies. The following cases were reported:

Case I. A female child, 5½ months old, was admitted to the Babies' Hospital on May 15th, 1897, with an entirely negative family history. The child's illness had begun one month previously with persistent vomiting. The head was of normal shape, and the fontanelles were not bulging. There was a soft spot over one parietal bone. The child had no teeth. On the fifth day after admission tremor and nystagmus developed. Nine days after admission there was a general convulsion, in which the mouth deviated to the left. Cyanosis was a feature of the convulsion. A second one occurred in ten hours. After these seizures the fontanelles were sunken. The child now became semi-comatose, and died after a few days. The autopsy showed the presence of hemorrhagic pachymeningitis, fibrino-purulent pleurisy, pulmonary congestion, fatty liver and nephritis. Along the superior longitudinal fissure, over the entire base and over the island of Reil on both sides was a membrane covering the pia. The ventricles were normal in size, and contained about one drachm of hemorrhagic fluid. There was fluid blood in all the sinuses. The cervical cord showed the same conditions. Under the microscope the right occipital region showed the pia attached to the cortex in many places, and there was a splitting up of the membrane overlying the cortex into two or more layers. The inner layer was infiltrated with small round cells. The outer membranous layers consisted of small round cells, fibroblasts and connective tissue fibres. The island of Reil showed the same condition, but much more marked, and about the same condition was present over the cerebellum. In the spinal cord there were only slight traces of hemorrhage.

Case II. Female infant, colored, 22 months old. The child had been nursed for seven months. It had never walked or stood alone, and was markedly rhachitic. The first two months in the hospital were marked by slight loss in weight and considerable prostration. In October, 1897, the child was re-admitted, with the statement that she had been well up to three days before, at which time she had had four convulsions, followed by three more the next day. The general condition was very bad. The hands and feet were in a position of persistent flexor con-

traction, characteristic of tetany. The knee-jerks were unobtainable; the fontanelles were bulging. There was slight but varying rigidity of the muscles of the back of the neck. Bloody mucous diarrhœa was present, and the child died in coma. The autopsy showed pachymeningitis hemorrhagica interna, broncho-pneumonia, and acute and chronic ulcerative colitis. Over the right side of the brain was a recent blood-clot covering the entire hemisphere, and over the left occipital lobe. The inner surface of the dura was covered with a membrane extending from the superior longitudinal fissure on either side. The pia was congested. The ventricles and brain substance were apparently normal. All the sinuses were filled with recent clots. The microscopical examination showed thickening of the pia over the right temporo-sphenoidal lobe, and the vessels of the pia were thickened. There was also a thick membrane splitting up into layers, as in the other case. There were numerous small blood vessels, and hemorrhage had occurred into the meshes of the membrane. In places, there were aggregations of small, round cells undergoing fragmentation. They were found chiefly in the superficial layers of the membrane. In the dura the fibres were separated from each other by serous infiltration, and the dura was covered with a membrane similar to that already described. In places, there was very extensive pachymeningitis.

It was at about five months of age, the speaker said, that this disease was especially frequent. The majority of these infants were badly nourished, many of them being subjects of rhachitis or of chronic colitis. The new membrane must be regarded as originating from proliferation of the dural endothelia cells. In some cases there was little inclination to hemorrhage. The membrane was very variable in thickness; sometimes it reached a thickness of two or three lines. It was especially prone to occur in the basal fossæ. There seemed no good reason for thinking that the locality of the pigmentation indicated that the layer of blood originated from the inner surface of the dura. On the other hand, there was no conclusive proof of the old notion that the disease was of inflammatory origin. It was so common to find severe intoxications without such lesions, that the intoxication theory did not seem to him tenable. It was apparently impossible to

recognize the condition until the hemorrhage occurred, and even then it was extremely difficult to make a positive diagnosis. Slight cerebral symptoms were probably masked in these very young and usually marantic children. The hemorrhage was probably more often unilateral, and the usual symptoms present were rigidity, hemorrhage and coma. Paralysis was rarely noted. The pyrexia was usually less than in meningitis, but these cases were so commonly complicated with other diseases that the range of temperature was very variable. He did not think there was any symptom or combination of symptoms in hemorrhagic internal pachymeningitis which might not be encountered in any acute infection without any cerebral affection being present; but whenever unilateral rigidity and convulsions, with deepening stupor, were present in a cachectic or rachitic child under one year of age, we should think of that diagnosis. It was probable that relatively slight traumatism to the head might occasion rupture of vessels in the highly vascular membrane. This gave these cases a certain medico-legal importance.

DISCUSSION.

Dr. Peterson remarked that the condition was interesting to him because of the possibility of its being found occasionally in infantile cerebral palsy.

Dr. Herter said that he was inclined to think that these membranes were considerably more frequent than one would suppose from the literature. It was quite possible to overlook the presence of the membrane if it were not decidedly vascular.

THE PATHOLOGY AND MORBID ANATOMY OF HUNTINGTON'S CHOREA, WITH REMARKS ON THE DEVELOPMENT AND TREATMENT OF THE DISEASE.

Dr. Joseph Collins said that the neurologist frequently encountered knotty problems, and among these none had the secret of its genesis more carefully concealed than the hereditary degenerative diseases. The pathogenesis of the acute inflammatory diseases of the nervous system was an open book, but the degenerative diseases were discouragingly slow in yielding the mystery of their being. This was especially true of such degenerative diseases as the hereditary ataxias, choreas and dystrophies. The status

of the original lesion could not always be inferred from a consideration of the lesion found at the time of death, and this was particularly true if the disease had existed a great number of years. No one could do much laboratory work on the central nervous system of individuals who had succumbed to degenerative nervous diseases of long duration without having forced upon him the fact that there are certain abnormalities of the circulatory system—varying degrees of degeneration of vessels, change in the size of the lymph spaces, and relative disproportion of glia tissue to the parenchyma—which occur with all degenerative diseases, considered entirely apart from their causation. He felt convinced that such changes were very often secondary, and had no other significance than as evidences of protracted disturbances of nutrition, and that this nutritional depravity was the result of the existence of the original lesion. There was nothing more certain than the occurrence of glia proliferation in all slowly progressing destructive lesions of the nervous system, but nothing could be more misleading than to consider this glia overgrowth to be primary, and the changes in the parenchyma secondary.

Huntington's chorea, Dr. Collins said, was a comparatively rare disease, and of rather recent recognition; hence the reports made upon its pathology had not been uniform. The discrepancies were apparently the resultant of the varying points of view of different observers. In studying the nervous system in cases of Huntington's chorea, it was scarcely justifiable to maintain that all the morbid conditions were inherent to the disease, for, as had been said, many of them might be the consequence of prolonged interference with nutrition. A study of two cases, he felt confident, would go far toward establishing the morbid anatomy hinting at the pathogenesis. His patient was a man, fifty-five years of age, who had married in early manhood, and who was the father of three children—all of them giving evidence of neuropathic inheritance. The known duration of the disease in his case was ten years. At the beginning the hands only were affected, but in the last years the lower extremities were also involved. The mind remained in fairly good condition up to about three years before his death, when he began to have suspicions about his relatives and friends, and became forgetful and

suicidal. His speech was so imperfect that in the last years of his life he was understood with difficulty. Dr. Collins had seen him for the first time a few days before his death. He then had a temperature of 105° F., and it remained at about this point until the end. The movements were very severe and incessant, except during sound sleep, although even then they frequently awakened him. He was quite conscious, but made no response to questions. The cause of death seemed to be exhaustion and high temperature. The disease was traceable to the maternal grandfather—an Irishman—who had three children, two of whom were affected with the disease. One of these was the mother of this patient, and of her seven children, five were afflicted with the disease. The other daughter had two children, one of whom became choreic. In three generations there had been no less than nine affected, and when it was considered that many of these children died in infancy, the number of cases that had developed was surprisingly great.

At the autopsy, on opening the skull, the dura was considerably adherent, the diploe dense and the Pacchionian depressions marked. The brain had a wet appearance, as did also the cord. The pia was not adherent to the brain. The convolution of the anterior portion of the brain were very small, and the entire encephalon weighed 43½ ounces. The dura was intimately adherent to the spinal column. The principal fissures were somewhat wider and shallower and shorter than in the normal brain, but there was nothing pointing to defective convolutions. The average thickness of the gray matter was uniformly less than in the normal brain cortex, but this thinness could not be attributed here to age. An examination of the pons and medulla oblongata did not show any marked variation from the normal, but the changes were more noticeable lower down. Microscopical changes were not confined exclusively to the Rolandic region, but the process here was more advanced. The specimens were stained by various methods, and carefully examined. The macroscopical changes were briefly as follows: (1) Thinness and atrophy of the cortex; (2) the mottled, streaked appearance and cribriform state on cross section of the brain in the fresh state, due to diminution in number and in health of the ganglion cells and to the increased peri-

vascular and pericellular spaces and increased patency of blood vessels. The microscopical changes were: (1) A decay or slowly progressive degeneration of the ganglion cells of the cortex throughout the brain, especially of the two deepest layers, the layers of large pyramids and polymorphous cells. This cell death was particularly evident in the Rolandic region, very much less so in the anterior pole of the brain, and incomparably less in the posterior pole. (2) Increase of glia tissue, but not sufficiently prominent to constitute sclerosis, the conspicuous increase being about blood vessels and ganglion cells. (3) Enlargement of the pericellular spaces and distention of the pericellular spaces. (4) Slightly diseased blood vessels consisting principally of a proliferation of the nuclei of the adventitia and a thickening of the intima. This involvement of the vessels was not regular or symmetrical, but showed itself in certain sections of vessels only. (5) Relative paucity of the medullated fibres of the cortex. In short, it might be said, that the lesion was a chronic parenchymatous degeneration of the cortex, with consecutive and secondary changes in the interstices, the brunt of the disease having been borne by the motor regions. There was, in consequence, a degeneration of the pyramidal tracts in the spinal cord. In Dr. Dana's case the central convolutions suffered most, and the process occurred in patches throughout the affected cortex. There was nothing to justify the opinion that it was an inflammation—the process was evidently one of degeneration.

In connection with the treatment, Dr. Collins said, that he desired to emphasize the necessity for delaying the advent of the disease in those who had a hereditary tendency to it, and also to emphasize the folly of tenotomy of the eye muscles—a method of treatment now being carried out upon one of these unfortunate individuals in this city, with a promise of a cure. If we wished to influence the cause of hereditary chorea after it had once become manifest, it would be necessary to administer whatever drug was selected in the largest possible doses consistent with life, and to maintain this medication for a long time.

DISCUSSION.

Dr. Onuf said that he had been present at the autopsy on the case reported in the paper, and had been especially im-

pressed with the general narrowing of the gyri—a general atrophy. On section, the mottled appearance of the cortex had been most striking, but the cribriform appearance produced by the enlargement of the perivascular spaces was also worthy of note. The general appearance of the brain resembled very closely that of a brain from a case of general paresis. The microscope confirmed the macroscopical appearances, although the changes were not as marked as one would have expected from the gross appearance. The cell changes were of the atrophic order. The characteristic feature was the accumulation of neuroglia cells in the pericellular and perivascular spaces. His impression was, that this accumulation was due to a secondary process following atrophy of the cells. The disease was evidently a degenerative one, originating in the parenchyma of the brain, and not in the interstitial tissue.

HEMIPLEGIA IN WHOOPING COUGH.

McKerron (*Brit. Med. Jour.*, Sept. 12th, 1896) reports the following: A child of five and three-quarter years, who had had whooping cough for three weeks, came into the house complaining of a headache and of feeling sick. She vomited once. Right hemiplegia and coma gradually came on, the latter continuing to grow deeper for three days, when it was complete. There were repeated right-sided convulsions. The condition remained stationary for three or four days, and then gradual improvement began, which progressed to almost complete recovery. At the end of three months the right leg was as good as the left, but the child preferred to use the left hand rather than the right.

PATRICK (Chicago).

UN NOUVEAU SIGNE D'EPILEPSIE (A New Sign of Epilepsy). Dr. Maïret. (*La France Méd.*, Jan. 29th, '97.)

Numerous experiments prove that the urine of epileptics is hypertoxic before, and hypotoxic after an attack. The hypotoxicity falls from 150 to 450 ccm. (the quantity of urine required to kill a rabbit of one kilogramme), and remains constant during the intervals between seizures. Hypotoxicity is also present in hysterical subjects, but the urine of epileptics gives rise to convulsive phenomena, whereas that of the former class is simply toxic. In all forms of epilepsy, typical, as well as epilepsy larvata, hypotoxicity exists, which is therefore so characteristic as to assume great clinical and medico-legal importance.

MACALESTER.

Periscope.

With the Assistance of the Following Collaborators:

CHAS. LEWIS ALLEN, M.D., Wash., D.C. R. K. MACALESTER, M.D., N.Y.
J. S. CHRISTISON, M.D., Chicago, Ill. J. K. MITCHELL, M.D., Phila., Pa.
A. FREEMAN, M.D., New York. H. PATRICK, M.D., Chicago, Ill.
S. E. JELLIFFE, M.D., New York. HENRY L. SHIVELY, M.D., N. Y.
WM. C. KRAUSS, M.D., Buffalo, N.Y. A. STERNE, M.D., Indianapolis.
W. M. LESZYNSKY, M.D., New York.

ANATOMY.

- I. THE STRUCTURE OF THE FIRST OR OUTERMOST LAYER OF THE CEREBRAL CORTEX. W. Bevan Lewis. (*Edinburgh Medical Journal*, vol i; 1897, p. 573.)

The studies whose results are given, were made on the brains of the rat, mouse, kitten, dog, sheep, ox, pig, the ape, and man. The methods used were the chrome-silver rapid method of Golgi-Cajal, the Weigert-Pal, and the author's "fresh method" with aniline blue-black. The "peripheral zone" as the author prefers to call this first layer, decreases in thickness, from the sagittal border of the hemisphere outwards and downwards, and also backwards. Its constituents are:

"First—Neuroglia and lymph connective elements.

"Second—Tangential or superficial medullated belt.

"Third—Terminal dendrites from the apices of pyramidal and other cells.

"Fourth—Termini of the second layer of nerve cells."

Of non-nervous cells, there are recognizable by the "fresh method," two kinds, one small, the other larger, and connected with a blood vessel by a peculiar, sucker-like process. By the silver method there appear several different looking cell forms, all of which however, are probably but different stages of the spider cell. An enormous number of medullated fibres run parallel to the surface, forming a layer whose depth varies in different parts of the cortex. These horizontal fibres are crossed by other fibres, vertical and oblique, making up a thick meshwork. There are also in this belt, numerous fine axis cylinders devoid of sheath.

The nerve cell processes come (1) from the pyramidal cells of the third and fourth layers and (2) from the cells of the second layer. The apical protoplasmic processes pass up towards the periphery, branching continuously so as to form a plumelike structure.

These processes are readily distinguished from the axis cylinders by their peculiar appearance due to the number of short processes they give off. The axis cylinders and collaterals are numerous, cross each other at various angles, and are in close relationship to the protoplasmic processes. Whether they arise mainly from the nerve cells occurring in this layer, is a disputed question. The author thinks that there are too few cells to account for them, and that most of them

come from the cells of the deeper layers. As to communication between them and the dendritic processes, he does not attempt to decide whether it is by continuity or by contiguity.

Going on, he describes the cells of the second layer. These present three peculiarities, great variation in contour, a very large nucleus, and a peculiar branching, giving the cell a somewhat horned appearance. There is sudden change in type of cells; in passing from one region to another, and a general suggestion of grouping of special cell forms, in particular regions of the cortex, in very specialized regions, one form occurring to complete exclusion of the others. These cells the author regards as probably sensory elements, and thinks that their varied form may have some connection with the varied qualities of sensation. In conclusion he suggests that the peripheral zone is an enormous field of the cortex, in which the transference of sensory currents into motor impulses is accomplished. This abstract gives but the briefest outline of this carefully prepared, and well illustrated article.

C. L. ALLEN.

2. LA NEOFORMATION DES CELLULES NERVEUSES DANS LE CERVEAU DU SINGE (New Formation of Nerve Cells in the Brains of Monkeys, Vitzou.) Archives de Physiologie, 9, 1897, p. 29.

An interesting series of experiments upon monkeys, carried on by Vitzou, seems to show that it is possible to reproduce new nervous tissue in the higher animals, although the contrary opinion has been held by many. His subjects were young monkeys. He removed portions of the cortex of the occipital lobes of both sides, thereby rendering the animals quite blind. But in the course of several months they began to regain their sight, and at the end of two years, could see fairly well. It was found upon repeating the operation, that a quantity of new nerve-like material had grown in the former site. It showed upon microscopical examination that nerve cells and nerve fibres were present. But after the second operation no new fibres grew, and the monkeys remained blind.

JELLIFFE.

3. ON THE REGENERATION OF PRE-GANGLIONIC AND OF POST-GANGLIONIC VISCERAL NERVE FIBRES. J. N. Langley. Journal of Physiology, 22, 1897, p. 215.

The author has conducted a series of experiments, chiefly upon cats, in which the cervical sympathetics were severed, the vagus being left intact. His general conclusions are as follows: The regeneration of pre-ganglionic fibres takes place by the formation of fresh terminations in connection with nerve cells. In nearly all cases, so far as regards the cervical sympathetic, the different classes of pre-ganglionic fibres form their new endings in connection with nerve cells of their own class and possibly in connection with the same nerve cells with which they were originally connected.

Nevertheless it appears that in certain conditions, pre-ganglionic nerve fibres are able to form endings in connection with nerve cells not belonging to their own class, so that, for example, pupillo-dilator nerve fibres, may become united during regeneration, with nerve cells which send their axones to the erector muscles of the hairs.

And it appears that the post-ganglionic nerve fibres ending in any one tissue can, if an opportunity be afforded them during regeneration, readily form nerve endings in connection with any other visceral tissue, so that for example pilo motor fibres may form nerve endings in the iris, and become pupillo-dilator fibres.

JELLIFFE.

PHYSIOLOGY.

4. LES CAUSES DES PARALYSIES RECURRENTIELLES. (Causes of Recurrent paralysis.) Lermoyez. *La Presse Medicale*, No. 36, '97.)

Considering first the pathology of the recurrent laryngeal nerve, the author formulates the following questions:

1. Does the recurrent contain centripetal fibres?
2. Ought the classic scheme for the distribution of the laryngeal nerves to be retained?
3. Does the recurrent laryngeal arise from the pneumogastric or from the spinal accessory?
4. What is the bulbar representation of the larynx?
5. What is the cerebral representation of the larynx?
6. Why do incomplete lesions of the recurrent fix the vocal cords in a median position?

These questions he answers as follows:

1. Negatively.
2. The classic scheme of distribution, while not explaining all observed pathological facts, should be preserved in default of a better one.
3. Is a question of interpretation. All are agreed that the larynx is innervated through nerve roots arising from the bulb in the region between the tenth and eleventh nerves, but some authors assign these roots to the pneumogastric, others consider them as belonging to the spinal accessory.
4. The larynx is represented in the bulb by two centres, one for respiration, the other for phonation, and each of these is bilateral. In the cat, the centre for respiration has been shown to be situated in the superior part of the floor of the fourth ventricle, while that for phonation is in the inferior part. Anatomical and pathological facts point to their similar location in man.
5. Respiration and phonation have most likely separate cortical representation. The cortical respiratory centre, though demonstrated by Russell in the cat and in the dog, is as yet not satisfactorily studied. The centre for phonation has been located by Krause in the dog, and by Horsley and Semon in the monkey; in the latter about the foot of the ascending frontal convolution. Each vocal cord is represented on both sides, and destruction of the centre on one side alone does not produce paralysis. In man there are recorded 4 cases which point to the foot of the ascending frontal as the seat of this centre, but they contradict in a measure the experimental observations, since lesion of one centre was followed by paralysis of the opposite vocal cord. The author does not think a definite conclusion can yet be drawn.

6. The median position of the vocal cord in partial lesion of the recurrent, is best explained by the theory of Semon, that the dilator muscles are inherently weaker than the constrictors, hence the first to be affected.

Passing to the location of the lesions producing the paralysis, the author studies their causation, in the recurrent itself, in the pneumogastric and spinal accessory nerves, in the medulla, and in the cerebrum. He concludes that, while paralysis of the recurrent is a serious symptom, it does not necessarily indicate a fatal issue, but must be considered in connection with the other symptoms present. He recognizes three principal types of recurrent laryngeal paralysis: 1. Grave and fatal. 2. Incurable but benign. 3. Curable and benign.

C. L. ALLEN.

5. UEBER ELEKTRISCHE REIZUNG DER ERSTEN DORSALWURZEL BEIM MENSCHEN (Electrical Stimulation of the First Dorsal Root). H. Oppenheim. Berlin klin. Wochenschrift, 33, 1896, p. 753.

In order to decide the question whether the oculo-pupillar fibres are in part derived from the first dorsal root or not, the author made use of a gunshot wound case in which the spinal canal was opened, exposing from the first to the fourth dorsal segment. Electrical irritation of the second dorsal root was negative; while immediately after irritation of the first, dilatation to a marked degree followed. This dilatation disappeared on removing the electrode. The author concludes, from his experiments, that the dilator pupillaris muscles receive their nerve supply from the first dorsal. JELLIFFE.

6. COMPARATIVE EFFECTS OF DIFFERENT ALCOHOLIC DRINKS ON MAN (Lanceraux.) Bull. Med. de Paris: 10, 1896, p. 979.

Lanceraux has investigated the changes taking place in the nervous system, due to abuse of different alcoholic drinks, wine, beer, absinthe, essences, etc.

In excessive use of alcoholic drinks of high percentage of alcohol, the tactile and thermal sensibilities do not seem to be greatly altered, while sensibility to pain seems exaggerated. In those that use absinthe and similar drinks to excess, the plantar reflexes are increased, light tickling causing movement, while slight stroking of the knees, legs, or abdomen causes pain severe enough to cause the patient to complain.

Similar results, although less marked, are to be observed in the upper extremities. In wine-drinkers, this sensitiveness of the skin is much less in the lower extremities; above there may be a zone of hyperesthesia; while still higher in the body, normal skin sensation is the rule. Psychical symptoms by absinthe drinkers are stated to be fewer than is generally supposed and taught in the ordinary textbooks. Wine and alcohol drinkers are prone to attacks of acute delirium, while in those that drink alcoholic essences, forms of dementia are more liable to follow. JELLIFFE.

PATHOLOGY.

7. BEITRAEGE ZUR KENNTNISS DER MULTIPLN, ALLGEMEINEN NEUROM (Contributions to a Knowledge of General Multiple Neuromata.) Karl Petré. Sonderabdruck aus dem Nordiskt Medicinskt Arkiv.

In this brochure the author presents an exhaustive study of a well-observed case of multiple general neuroma. The histological work was done at the Pathological Institute of the University of Lund. A complete review of the literature is given. The patient was a tailor, aged thirty-two. There was a high degree of neuro-pathological hereditary taint, but not as is generally the case, of a type similar to the disease. The patient's father was a man of good intelligence, but of violent temper, and immoderately addicted to alcohol. A paternal uncle was also intemperate. The mother was eccentric and of low degree of intelligence. One of her sisters was insane. A brother and sister of the patient was feeble-minded, and another brother actually insane. A maternal aunt and cousin were at times under treatment for mental disease. The tumors were very numerous and widely distributed over the trunk and limbs, corresponding to the distribution of the nerves affected. They varied in size from a pea to a pigeon's egg and could be distinctly felt under the skin. Where multiple neuromata do not undergo sarcomatous degeneration, there have generally been no special nerve symptoms observed. Here, however, these

tumors caused widespread motor and sensory disturbances of the same kind as are occasioned by a very chronic and benign neuritis. Slight ptosis, nystagmus, inequality of the pupils, sluggish reaction for light and accommodation, diminished tactile sensibility, and temperature sense were observed. There was a considerable paresis of the left arm, and the reflexes of the arm were distinctly increased. Motion both in the upper and lower extremities was distinctly ataxic, and the "muscle sense" showed considerable diminution. Romberg's symptom was present. Patellar and plantar reflexes were increased. The expression of his face and conversation evidenced distinct mental impairment.

In the case here presented there was in all probability compression of the spinal cord due to tumor formation on the roots of origin of the spinal nerves. Cases of such compression have been observed by Sibley, Riesenfeldt, Gerhardt and Sieveking. The oculo-pupillary disturbances have been observed but twice before and have been reported by Riesenfeldt and Herczel. Their cause is not known with certainty. The clinical picture corresponds very closely to that occurring in neuritis of the Dejerine type, except that in the latter the symptoms are all more strongly developed.

The most important feature of the microscopical examination of the excised neuromata was the demonstration of a new formation of non-medullated nerve fibres which the author has shown for the first time. It is in entire harmony with the clinical symptoms that a considerable destruction of nerve fibres should be found only in those cases which undergo sarcomatous degeneration. In other cases there are sometimes no changes whatever in the nerve fibres, sometimes there are slowly developed alterations in these elements, generally a pressure atrophy, which is also accompanied by a diminution in their number. In the cases here reported there was a very slow but undoubted destruction of nerve fibres which corresponded fairly to the clinical manifestations. In a second case of multiple neuroma the author succeeded in demonstrating in all probability a new formation of medullated nerve fibres. The appearances corresponded to those which are observed in the proximal stump of a divided nerve when regeneration occurs. It is not possible to say whether or not this reproduction of nerve fibres is throughout more extensive than is ever the case in neuromata and can even go on to complete disappearance. In the former the place of the nerve fibres is taken by a fibrous connective tissue, and in neuromata one often finds almost the same form of connective tissue surrounding the bundles of nerve fibres which pass through the centre of the tumor. In the latter, however, there is also diffuse connective tissue that forms the principal mass of the tumors. In multiple neuroma as in the above-mentioned neuritis there is often a neuropathic taint which in both may assume the form of a similar heredity. Both diseases are generally congenital or appear in childhood and are to be attributed to faulty development. In both this probably consists in an increased energy of growth of the connective tissue of the nerves, or in a relative diminution of the same for the nerve fibres. As regards neuromata, one can agree with Goldman that this faulty development is to be regarded as retarded growth, that is, a persistence of the connective tissue of the nerves in the embryonal stage.

General multiple neuroma and neuritis of the Dejerine type are to be considered in all probability as related diseases. When one compares these two diseases which are produced by increase in the connective tissue of the nerves, the thought occurs that the development of tumors may be regarded as a safety valve for the exuberance of

connective tissue, and may indeed be a conservative process which in certain cases protects with varying effectiveness the nerve fibres from destruction. In neuritis of the Dejerine type is to found a premonition even if a slight one, of tumor formation. SHIVELY.

8. LESIONS DE LA MOELLE EPINIERE DANS UN CAS D' AMPUTATION CONGENITALE DES DOIGTS. (Lesions of the Spinal Cord in a Case of Congenital Amputation of the Fingers.) Longues et Marinesco La Presse Medicale, No. 45, 1897.

After giving a short description of a case of congenital absence of the first and middle finger, with rudimentary ring finger and deformity of thumb and little finger of the right hand in a woman who died at 63 of a cancer, the authors give the result of the histological examination of the spinal cord from that patient. The cervical cord from the first dorsal segment to the medulla was examined. There was diminution in size of the right half of the cord, most marked about the eighth cervical and first dorsal segments, and progressively less marked towards the higher level. On the right side both anterior and posterior roots were diminished in volume, and the reflexo-motor collaterals which traverse the posterior horn to go to the motor cells of the anterior horn, as well as the collaterals going to cells of the posterior horn were reduced in number, the change being most pronounced in the lowest segments, but visible as high as the fifth cervical. In the right anterior horn the changes were quite marked. It was smaller and paler than the left, and its cells showed changes: the antero-internal group being well preserved, the postero-lateral group partly atrophied, while the median group had disappeared entirely. The cells of the gray matter between the anterior and posterior horns were reduced in numbers. Above the fifth cervical these changes were no longer visible. There was an atrophy of the column of Burdach, most marked below, but traceable up to the nuclei in the medulla. The columns of Goll showed degeneration on both sides, but the authors do not think this explained by the lesion of the hand. The paper closes with a consideration of the origin of the disease process, which the authors conclude to be exogenous and secondary to the amputation in utero of the fingers. The changes found confirm the views with regard to the distribution of the collaterals of the posterior root fibres, held by Kolliker, Ramon y Cajal and others. C. L. ALLEN.

9. UEBER VERAENDERUNGEN DES MENSCHLICHEN RUCKENMARKS NACH WEGFALL GROSSERER GLIEDMAASEN. (Concerning Changes in the Human Spinal Cord after Removal of Large Portions of the Limbs.) By E. Flatau. Deutsche med. Wochenschrift, 23, 1897, p. 278.

Flatau had the opportunity of examining the spinal cord in two cases a short time after amputation had been performed. He found that the cells in the parts corresponding to the amputated limbs, when stained by the method of Nissl, were enlarged and tubular, and that the protoplasmic processes were decreased in number. The chromophilic elements had undergone a gradual change, and the nucleus in some cells was eccentrically situated. These findings correspond with those obtained experimentally. He states an important fact, acknowledged by many, viz., that it is possible to detect not only alteration of the cell from the action of different substances, but also differences in the action of the various substances on one and the same cell species. He found both the anterior and the posterior roots degenerated after amputation. [There is a suggestion in this statement in regard to the possibility of a peripheral origin of tabes.] SPILLER.

10. PARALYSIE BULBAIRE ASTHENIQUE DESCENDANTE, AVEC AUTOPSIE, (Descending Asthenic Bulbar Paralysis, with Autopsy.) (Symptom complex of Erb.) *Bulletins et Mémoires de la Société Médicale des Hospitiaux de Paris.* By Widal and Marinesco, 14, 1897, p. 518.

The writers give a succinct statement of the symptoms of this disease, and show, by a very important case of their own, that, contrary to the opinion of Goldflam, the disease may be acute and death occur within a short time.

A tuberculous man began to complain of headache. This was followed by unilateral, and later bilateral, ptosis, partial bilateral facial paralysis, noticed especially in the distribution of the upper branch of the right seventh nerve, difficulty of speech, mastication and deglutition, convergent strabismus in both eyes, rapid exhaustion of certain ocular muscles, paralysis of the tongue and muscles of the neck, and weakness in the upper extremities. There was no positive disturbance of objective sensibility, and no muscular atrophy. The symptoms varied from time to time, and were less severe in the morning. Death from suffocation occurred about three weeks after the beginning of the headache, and about two weeks after the first sign of paralysis (ptosis). The unusual symptoms in this case were paralysis of the external recti, partial paralysis of the muscles supplied by the third nerves, and paresis of the iris.

The histological examination was made by the methods of Nissl, Marchi and Pal, and lesions were found by the first two, consisting of disintegration of the chromophilic elements in the nuclei of the cranial and cervical nerves, and degeneration of the myelin sheaths in the third, seventh and twelfth nerves. These were not post-mortem changes, and probably were not due to the tuberculous condition of the patient. The intensity of the cellular lesions was proportionate to the intensity of the symptoms. There was nothing peculiar in the alterations of the chromophilic elements; they were such as are produced by intoxication, and the asthenic paralysis may be the result of an intoxication, endogenic or exogenic.

Widal and Marinesco are the first to report cellular changes, independent of perivascular lesions and interstitial inflammation, in asthenic bulbar paralysis. They believe that this disease and the polioencephalomyelitis may be due to nuclear lesions, though of different nature and degree, and that the two processes should be regarded as distinct. It does not follow from one examination that the symptom-complex of Erb is always the result of similar lesions. SPILLER.

CLINICAL NEUROLOGY.

11. VERTIGO AND OCULAR DEFECTS. W. Osler, *Montreal Medical Journal*, 25, p. 12.

The author, discussing the association of vertigo and ocular defects, cites a case of the former which was of the most intense character, and had persisted for eighteen months, but which was completely relieved by properly adjusted glasses.

Mr. H., aged 54, consulted the doctor on the 4th of April, 1894, complaining of vertigo and stomach trouble. He has been a healthy man with the exception of attacks of biliary colic. The patient was a brick maker by occupation. His habits have been good. He has been a steady smoker until about a month ago.

For about eighteen months he has had attacks of severe vertigo associated with flatulency. The first one occurred while he was sitting at the table in a restaurant drinking claret punch. He jumped up and said to his wife, "Catch me, catch me," and had to get hold of the table to steady himself. He had a sensation as if a cannon-ball had

burst in his head, and as if everything was in motion. The attack lasted about an hour. He did not vomit but looked pale, and broke into a profuse perspiration. He has had only two attacks of similar severity, one while in his carriage. He said it seemed as if the horse was down and everything was turning over. This attack lasted about an hour. He had to go to bed, and felt very badly, and after it he felt confused in his head.

The milder attacks had occurred with greater frequency. Scarcely a day passes without one or two; thus, one day after breakfast his stomach felt badly, and he had a good deal of belching. Then, as he expressed it, his head went off at once, and he generally cried to his wife, "come and catch me!" Coming home just before dinner he had another spell. When they are at all severe, he gets pale and cool, and perspiration rolls off his face in beads. He belches all the time during an attack, and on some days he belches continually. He had no pain whatever in the chest or elsewhere. The attacks did not come on during sleep, but he had several of them while in bed.

From his statement the vertigo was apparently both subjective and objective. Objects went to the right, but he felt that he turned also. While the attack lasted walking was impossible. If the head were held tight, the attacks did not appear to be so severe. Though the patient fainted, yet consciousness was always preserved. There was no throbbing at the heart. The longest interval the man ever passed without an attack was two weeks.

The patient attributes his condition principally to his stomach, as the distress and the belching were incessant.

Though he did not complain of difficult hearing, it was evident that he was a little deaf, and on questioning him, he stated that the deafness had been coming on for several years past, particularly in the right ear in which there is a ringing noise almost constantly. In the spells it is much lower, and sometimes there is an explosive burst.

On examination there was found deafness in the right ear, due to changes in the auditory nerve or its expansion in the labyrinth, and that there was slight deafness in the left ear. The examination of the eyes showed a rather high grade of hypermetropia with a decided amount of astigmatism.

The change in the patient from the use of properly adjusted glasses was most remarkable. The severe, as the mild attacks, wholly disappeared, and even his stomach troubled him less. Two months after the patient suffered a severe attack of uncontrollable vomiting which ended fatally. The autopsy revealed an acutely developing malignant disease of the stomach.

Dr. Osler concludes this interesting report with the following suggestive remarks: A condition of irritation and instability of the space nerve centres may possibly be kept up by serious accommodation errors. Physiological, clinical, and well-established anatomical data show the association between the labyrinth and the oculo-motor mechanism. The case which was reported bears on a practical aspect of the question, inasmuch as the patient obtained complete relief from a vertigo of the most intense and persistent character by the use of carefully adjusted glasses.

ABRAHAMS.

12. FRIEDREICH'S ATAXIA. E. Riggs: Northwestern Lancet, 16, 1896, p. 264.

The author reports two cases. The first patient, a boy, aged 16 years, had had difficulty in walking for a year, which had rapidly increased before the date of examination. He had some difficulty in articulation, the gait was spastic and staggering, all voluntary move-

ments were very slow, and he had had two attacks of profuse sweating without apparent cause. There was slight transverse nystagmus, ankle clonus and rectus clonus on both sides, but the knee jerk was absent (!). There was also well-marked gluteal clonus.

The second case was a girl, nine years old, who had suffered from increasing ataxia for some months. A brother had died of a similar affection at the age of twenty years. She had transverse nystagmus, but the pupil reflexes were normal. As in case one there was a well-marked lateral curvature of the spine. It might be noted that twitching of the head and neck was present, and tremor of the hands accompanying voluntary effort. There was well-marked ataxia, the superficial reflexes were present, but the knee-jerks were absent.

PATRICK.

13. FRIEDREICH'S DISEASE. Moyet: *Northwestern Lancet*, 17, 1897, No. 2.

The author reports two brothers, aged respectively 16 and 14, both of healthy parents, affected with Friedreich's disease. Reinhold (16 years) has it for four years, and Carl, the younger one, for two years. In Reinhold the affection followed an attack of diphtheria, and in Carl it developed without any preceding illness. The ataxia was much aggravated in the latter after a fracture of the leg. The symptoms in both boys are identical save, perhaps, that they are a little less severe in the younger child. They are as follows: the feet in walking are placed widely apart and the toes somewhat inverted. The gait is shuffling and unsteady with some swaying of the body. Cannot walk a straight line. Sudden turnings produces staggering and falling. If unsupported, will fall when eyes are closed. Ataxia is very marked in both hands. No sensory disturbances; no trunk anesthesia; no nystagmus nor muscular tremor. The pupils and eye-grounds normal; knee jerk absent: no pain or paresthesia. All the rest of the children in the family, six in number, enjoy good health.

ABRAHAMSON.

14. THREE CASES OF FRIEDREICH'S DISEASE WITH INCREASED KNEE-JERKS. (Hodge, *Brit. Med. Jour.*, June 1897, p. 1,405.)

The three patients, two sisters and a brother, aged 44, 40 and 39 years respectively, presented practically the same clinical history and condition. About the age of 12 they began to have difficulty in walking, which disability gradually increased and ultimately the typical club-foot of Friedreich's disease developed. All were found to have marked incoordination, nystagmus and speech defect, but the knee-jerks were distinctly exaggerated.

French authors would probably class these cases as hereditary cerebellar ataxia.

PATRICK.

15. MALADIE DE FRIEDREICH A DEBUT TARDIF. (Friedreich's Disease with Late Onset.) Gaston Bonnus. *Bulletins de la Societe Anatomique de Paris*, 10, 1897, p. 18.

The author presented a case of Friedreich's disease with notes upon the autopsy and microscopical findings.

The case occurred in a man thirty-nine years of age, although the earliest signs came on about his fourteenth year.

The hereditary factors were marked, one sister being markedly ataxic in the lower extremities without sensory disturbances and a brother of thirty-one had had typical signs of the disease for six or seven years.

The results of the histological examination is of interest by reason of the rarity of such examinations.

The cerebellum was said to be absolutely intact.

Spinal Cord: At level of third lumbar, the posterior columns were degenerated save in the region of Flechsig's centrum ovale and Westphal's zone. A certain amount of degeneration was found in the crossed pyramidal tracts.

Ninth Dorsal: Here the posterior columns were markedly degenerated. The crossed pyramidal tracts were degenerated, also Gowers bundle and the direct cerebellar tract to a less extent. The cells of Clark's columns were somewhat atrophied and were fewer in number.

Eighth Cervical: The entire peripheral region was involved, but the degeneration was most apparent in the columns of Turck, and Goll, and Burdach.

Medulla: The sensory columns were involved as high as the nucleus gracilis and cuneatus, but the degeneration did not extend beyond these nuclei.

The meninges were intact, the anterior roots were normal, but the posterior roots were markedly degenerated. The median, ant. tibial, sciatic and musculo cutaneous nerves were affected.

JELLIFFE.

16. UEBER FRIEDREICHSCHE KRANKHEIT (Hereditäre Ataxia). Concerning Friedreich's Disease (Hereditary Ataxia). G. Rosenbaum. Deutsche med. Wochenschrift, 22., 1896, p. 471.

Two children respectively 10 and 13 years old, showed the first symptoms of this disease 6 years ago. A brother of 15 is healthy, as are also the closely related parents, the father's two brothers and a sister, and a cousin of the mother died of diabetes. The first symptoms appeared in the elder girl soon after an attack of whooping-cough, the younger showed signs of the affection shortly after. A further exposition of their present status is unnecessary, as both cases present the typical picture of pure Friedreich's Ataxia.

Pierre Marie differentiated a particular form of the original Friedreich's disease, the so-called "cerebellar hereditary ataxia," in which the lesion is to be sought in the cerebellum, while spinal changes are either altogether absent or so slight as to be inappreciable. The characteristics differentiating this form from the true Friedreich's ataxia are its later manifestation, (20th, 30th year), the fact that the upper extremities are not attacked until the disease is well advanced, and that patellar reflexes are either normal or above normal, and the absence of sensory disturbances and scoliosis. On the other hand, visual disturbances, impaired sensitiveness of the pupil, etc., are invariably present. Although transition forms are unquestionably to be found, the author thinks that spinal and cerebellar sub-groups should be introduced under the general heading of hereditary ataxia.

VOGEL.

17. PARAPLEGIE SPASMODIQUE FAMILIALE. (Hereditary Spasmodic Paraplegia.) C. Achard et H. Fressonn. Gaz. hebdom. de med. et de Chir., 1, 1896, p. 1225.

Two cases of hereditary spastic spinal paralysis affecting sisters, are reported. Although as a rule this disease manifests itself between the ages of 3 to 12 years, or still later, in the present instance the first symptoms were remarked in both children at the time when they commenced to walk. In the one case it was at the age of 16 months, after an attack of some acute disease, the nature of which it was impossible to learn about definitely, in the other at the 12th month, after an attack of smallpox, that the disturbances first took place. The disease was remarkable for the slowness of its course, although steadily increasing in severity. Both parents were entirely sound, but a sister of the patient had an epileptic history.

JELLIFFE.

18. HEREDITARY ATAXIA—FRIEDREICH'S DISEASE, D. Brower; Journal of the American Medical Association, 28, 1897, p. 871.

Three cases of Friedreich's disease are reported from the clinic of the author. All the cases, two boys and a girl, were of the same family, and all exhibited marked stigmata of degeneration. The symptoms observed were:—1st, the ataxia beginning in the lower limbs and extending to the arms and tongue; 2d, the spinal curvature, double scoliosis; 3d, gradual development of paraplegia; 4th, loss of knee-jerk; 5th, the development of the condition in childhood; 6th, the absence of Argyll-Robertson pupil, of anaesthesia, of vesical symptoms, of severe pains, of intention tremor, of spasticity of the gait. These several symptoms readily differentiate the disease from posterior spinal sclerosis and multiple sclerosis. The seat of the morbid changes is at first the posterior and lateral columns of the cord, later the anterior cornua and finally the nerve structure. In some cases there is developmental failure; in others, an inflammation resulting in a steady encroachment of the connective tissue on the nerve elements.

SHIVELY.

PSYCHIATRY.

19. ACUTE DELIRIUM.

Two recent contributions to the subject of acute delirium deserve notice. Coston (Nashville Jour. of Med. and Surg., Aug., 1896) defines it as "a very acute febrile disease of the brain, usually fatal, attended by wild delirium, hallucinations, and great disturbance of motor functions." The cause is obscure, writings on the etiology being scarcely more than speculations. It affects both sexes, is more frequent under 30, and apparently bears no relation to heredity. The onset is usually sudden and the first symptoms mental. Three cases are detailed.

Case I. A girl of 15, with unimportant family and negative personal history was, when first seen, "restless and slightly delirious, using uncouth language and complaining of pain in her head and also of pains in her hips and back; this latter seemed but insignificant in character, but she complained bitterly of the headache. There was great motor excitability, the patient desiring to be in constant motion, and occasionally cursing and abusing those about her." Morphine and bromides failed to quiet her, and the next day she was rather worse; temperature, 101; pulse, 100; and she constantly talked irrationally, using the foulest language, but recognized everyone. There was absolute anorexia, no vomiting, and the urine was normal. She was very destructive. After 30 grains of chloral she slept five hours, but awoke more restless and destructive than before. Fifteen grains of the same drug induced comparative quiet, but she was soon as bad as ever. Sulphonal, trional, paraldehyde were tried, but large doses of hyoscine hydrobromate were most successful. 1/20 grain producing six to eight hours sleep. She gradually became weaker and, *pari-passu*, the delirium less violent, and died on the thirteenth day. The highest temperature was 102, the lowest 100.5, and for a few days preceding death she seemed to be blind; the pupils were widely dilated.

Case II. was a girl of 13, with good family and negative personal wildly delirious, violently and constantly agitated, very profane, who had never menstruated, but showed signs of approaching puberty. The character of onset is not stated. When first seen she complained bitterly of headache. The temperature was 100.5; pulse, 110. It was thought at first to be hysteria, but she did not improve and died suddenly in the beginning of the fifth day while sitting on a commode. There was no autopsy in either of these cases.

Case III. A stout country girl, aged 20, of good heredity, and

with unimportant personal history, was taken, while at church, with what was thought to be a chill, but the shaking tremor and slight delirium continued, and she was seen on the third by the reporter. She recognized acquaintances but "drifted into all kinds of foolish talk," and could not remember having seen a person a few minutes before; temperature, 101; pulse, 100. The body and extremities were in constant motion. The physical agitation was in some degree controlled by morphine, but it had no effect on the delirium unless it were to make it worse. She continued *in statu quo*, except that she lost weight rapidly. She was given sedatives and hypnotics, but hydrobromate of hyoscine, never less than 1/50 grain, produced the best results. At the end of the week the physical agitation was somewhat better, but she continued to lose strength for about three days longer, the temperature varying from 100 to 103, with pulse from 100 to 150. At this time she was found to be totally blind, though hearing and smell remained very acute. The amaurosis lasted four days and then gradually improved. Mental improvement began a week later than the physical betterment, both gradual, and so continued until complete recovery, with the exception of one slight relapse; but there was still some jerking, especially of the right arm and hand under excitement.

The author calls special attention to the great physical agitation, rapid exhaustion, hyper-acuity of hearing and smell, the occurrence of blindness and the vesical weakness which often required the use of a catheter. The patient recognized persons, but talked irrationally and forgot immediately having seen one.

As to differential diagnosis from acute mania, the author has the following to say:

"Acute delirium and acute mania are more frequently mistaken for each other, and their diagnosis from each other is more difficult to make, but with care we may differentiate them. The symptoms of acute delirium are much graver, the course briefer and more definite, the temperature is elevated in acute delirium and lowered in mania; the exhaustion is very rapid in acute delirium, while the maniac will continue to rave for months with little perceptible loss of strength; mania is a conscious delirium, the patient being aware of what he is doing and taking every advantage of you; acute delirium is an unconscious delirium, the patient never trying to take any advantage of you, and although he recognizes you, five minutes later he does not remember to have spoken to you. In mania the appetite is often enormous; in acute delirium it is always absent; mania is preceded by marked prodromata; the prodromata of acute delirium are never very marked and often absent; in mania the face is often flushed and the sclerotic injected; in acute delirium the face is pallid and no injection of the sclerotic; acute delirium will terminate in death or recovery in two or three weeks, mania will require months."

Babcock (Medical Record, Aug. 1st, 1896) first calls attention to the very great discrepancy in the percentage of cases of acute delirium admitted to different asylums for the insane. This varies from about one-seventh of one per cent. to 4.7 per cent., which enormous difference the author rationally attributes to the diagnostic tendency of individual institutions. The paper is essentially devoted to a consideration of the nature of acute delirium, particularly its possible bacterial origin. The author accepts the classification of Wood into acute periencephalitis, and cases in which no lesion can be demonstrated, but in which the affection is assumed to be one primarily of the cortical cells. The case reported is placed in the first of these two categories. The patient was a man, 46 years of age, of good family history, but addicted to the excessive use of alcohol and tobacco. Ten days before admission he became restless, sleepless and talkative, and later was at

times violent. On admission he talked almost constantly and entirely incoherently, was physically agitated, and it was impossible to attract his attention. The temperature was 99.6; pulse, 80; urine practically normal. Patellar reflexes were absent. After the administration of 20 grains of sulphonal, he slept seven hours and awoke with no temperature, but as delirious as before. He continued in much the same condition, the temperature being usually normal, until the twenty-second day of the disease, when the delirium increased. The temperature arose to 100.2, pulse to 100, and a trace of albumin appeared in the urine. Sulphonal failed to produce sleep and hyoscine was substituted with good results. He remained in the same condition till the twenty-ninth day when lumbar puncture was performed with apparently some transitory relief. The patient then gradually grew weaker, passed into a typhoid state, and died on the forty-sixth day of the disease, greatly emaciated. Eight minutes after death spinal puncture was again done, and 66 c.c. of turbid fluid collected. The post-mortem examination showed macroscopic and microscopic signs of inflammation in the membranes of the brain and in the cervical cortex. The fluid from the first puncture contained 2/25 per cent. of albumin, and that removed after death 3/5, which is exceedingly high and would indicate acute inflammation. Bacteriologic examination of the fluid showed the micrococcus of pneumonia and streptococci. Inoculation of rabbits caused symptoms of infection, but the experiment was not conclusive.

PATRICK.

THERAPY.

20. TRAITEMENT DES DOULEURS DE L' ATAXIE PAR LE BLEU DE METHYLENE (Ataxia Pains Treated by Methylen Blue), M. G. Lemoine, *Gazette Hebdomadaire*, 2, 1897, p. 570.

The author reported the use of methylen blue in nine cases of ataxic pain. In two there was no improvement; in five there was a great lessening of the intensity and frequency of the crisis, and in two a long period of complete ease. The author believes that the pains which yield the most readily are the severe lancinating pains in the legs, and girdle pains; the most difficult, those which are seated in the intestines, the stomach or the gastric crises without other pains. The effect sometimes lasted for days, and occasionally for weeks. Methylen blue seems to have a wide range of usefulness, if the therapeutic optimism of the French observers does not mislead them; besides the valuable property of relieving ataxic pains, diabetic symptoms have disappeared under its use, and even the final elimination of sugar from the urine has been ascribed to it. It is indeed almost too useful.

MITCHELL.

21. PATHOGENESE UND BEHANDLUNG DER CHOREA, (Pathology and Treatment of Chorea). P. Cheron. *Allg. Wiener Zeitung*, 41, 1896, p. 429.

Chéron recommends methodically used massage, which he claims to have used with much success. Massage and passive movements should be employed several times daily, together with cold baths, cold douches, cold packs, and notably cold, violent sprays against the vertebral column of a temperature of from 8 to 10 deg. C. for about 1/4 to 1/2 minute. Heart complications form no contra-indication to the use of cold water, as children usually have few signs of circulatory difficulty. But the patients should never be frightened by hydiatic measures, else the psychic excitement, restlessness and pathologic movements become increased. The heart should be examined daily, and its condition forms a guide to the choice of whatever form of cold-therapy seems indicated.

STERNE.

22. L'ANTIPIRINA CONTRO LA COREA, (Antipyrin in Chorea Minor). Negro. Gaz. deg. ospedalé, 17, 1896, p. 308.

This drug was exhibited with marked success in chorea. Dose for children of from five to seven years, 0.8 to 1.0 gm. pro die: from eight to ten years, up to 2.0 gm. Older children he gave up to 3.0 gm. pro die. The drug was usually readily taken, with few ill effects, if a pause of from 3 to 4 days was allowed to follow the day upon which antipyrin was used. His formula is

R Antipyrin	10.0
Aquae.....	200.0
Syr. Cort. Aurant.....	50.0
M. D. Sig. as directed.	

The antipyrin was given for about one month, and was followed by arsenic and iron until complete cure resulted.

STERNE.

23. TREATMENT OF NEURASTHENIA BY PSYCHOTHERAPY. Dr. Valentine (Medical Week, July 31st, 1896).

The extent to which physicians⁸ possessed of one idea will go is illustrated in the report of the remarks of Dr. Valentine at a meeting of the Hypnological and Psychological Society, July 20th, 1896, at Paris. He describes the treatment by hypnotic suggestion of three patients, only one of whom really, according to his own description, was in deep sleep; the third case, he says, was treated for three months by "suggestion without hypnotization." This apparently means that he encouraged the patient and told her she would get well. Other physicians, who do not pretend to hypnotize their patients, have no doubt done the same thing, without knowing that they were qualified for members of the society with the imposing name. MITCHELL

24. TREATMENT OF THE INSOMNIA OF INSANITY BY TRIONAL AND TETRONAL.

M. A. Bohn (Thèse de Paris, 1896) in a study of the treatment of the insomnia of insanity by trional and tetronal, concludes that both drugs are powerful hypnotics and that their actions are practically identical. He thinks that either is preferable to chloralose on account of the accidents which occasionally follow its employment, and on account of the wide difference in doses required by different patients. [The reporter has twice seen very alarming symptoms; prolonged unconsciousness, breathing as slow as if the patient had taken a large dose of opium, fixed pupils half dilated and mental confusion for some hours on waking, as the result of the use of 15 grains of chloralose in one neurasthenic case and of 10 grains in another.] MITCHELL.

25. SEDATIVES AND HYPNOTICS IN THE TREATMENT OF INSANITY. Discussion before the British Medical Association (British Medical Journal, Sept. 26th, 1896, p. 807.)

Oswald regarded failure of strength as the most important symptom making it urgent to procure sleep in acute cases. In such cases he preferred paraldehyde; it did not depress and had no bad effect. He thought in the rush after new remedies that old and meritorious ones were apt to be forgotten, and instanced as such conium juice, digitalis, and chloral, especially when combined with bromide. This last drug, combined with hyoscyamus and cannabis indica, could be given in much larger doses than were usually employed. He was opposed to the use of sulphonal, as it, in many cases, destroyed the red blood corpuscles and produced mild dementia. It cut short periods of excitement, but the patient did not regain mental clearness. Morphine was not so useful as opium, and his impression of hyoscyne and

hyoscyamine was not favorable. The best hypnotics and sedatives, he thought, were exercise, work, distraction of thoughts, amusements, etc.

J. A. Campbell thought that hypnotics and sedatives had no curative influence, and that the continued use of such drugs for any length of time had a tendency to retard recovery.

McLeod said that in discussing such subjects there was usually too much generalization. Such drugs should be given not to any class of disease, but with a strict attention to the individual and his constitutional peculiarities. Sulphonal had a distinct appetizing effect. He never saw any real harm result from its proper use. It usually did harm in cases of circular insanity. It might shorten the excitement, but it prolonged the depression and clouded the quiescent period. He used fewer drugs of that class than he used to do. The most efficient hypnotic he had found was a solution composed of chloral hydrate, potassium bromide, hyoscyamus and cannabis indica. He had used no preparation of opium as a sedative, only as an anodyne. Hyoscyamine was occasionally of use, and in moderate doses allayed the restlessness of certain organic cases, such as those of brain tumor, more effectually than any other drug.

Yellowlees summed up his convictions regarding hypnotics in the phrase "The less the better." There should be a definite distinction drawn between the extent to which such drugs were given in incurable cases and in cases in which there was a reason to believe that recovery might take place. We should put up with a great deal from a curable patient before we ran the risk of the brain cell injury which these drugs were so apt to inflict. They always retarded and sometimes even prevented recovery.

McDowall said that he used to give hypnotics very freely, but now very rarely indeed. He still uses them extensively in one class of cases, however, namely melancholia. He had been told by patients that physicians did not properly understand the great misery of sleeplessness. To diminish this he gave his melancholiacs chloral, sulphonal or other hypnotic. He never used such drugs in cases of mental excitement. He practically never gave morphine or hyosine.

Carlyle Johnstone said that in prescribing sedatives and hypnotics in the treatment of insanity he would be guided by the same general principles as those which would guide him in treating other diseases. He would ask himself, in the first place, is a sedative or hypnotic really required in this particular case? Is it necessary that the patient should be put to sleep or that his excitement should be suppressed? Is our object being attained? Has sleep been obtained? Has quietness been procured? If our object has been attained, has it been attained at too great a cost? In a word, is the patient the better or the worse in body and mind? If the drug was interfering with any natural function, if recovery was not being promoted under its use, if the patient could not be said to be better than he was before, he would say that, even although sleep and quietness had been produced, they failed in their purpose.

Gairdner condemned opium and stimulants in the treatment of delirium tremens.

Urquhart did not see that it was permissible that a chronic incurable melancholiac should be left in misery day and night without some relief being given him. Certainly great care was necessary in the use of hypnotics. The moral of the discussion seemed to him to be "be-ware."

Turnbull said he thought these drugs were very useful in tiding over an emergency. He had found sulphonal most useful, and he had never seen any ill effects from it.

Douglas said that the most valuable use of hypnotics was to be found in that period which preceded the state in which a patient could be certified. They could often tide over a crisis at such a time by means of drugs. He had found croton chloral and bromide of the greatest value. He also recommended tetronal in 10-grain doses as a hypnotic.

PATRICK.

26. SATURNINE ENCEPHALOPATHY TREATED BY VENESECTION AND SERUM INJECTION.

M. Desplats (*Journal de Médecine*, Nov. 10th, 1896) recommends the injection of serum associated with bleeding in this trouble, and mentions the case of a man who had had two attacks of colic, followed by an epileptiform crisis characteristic of saturnine encephalopathy. The urine contained no albumen. After the patient had been bled, 600 cubic centimetres of artificial serum was injected subcutaneously. There were no further attacks, and in a few days the case was discharged cured. The author considers it legitimate to attribute the cure, in this instance, to the association of the two means, which he says, have never before been employed in eclampsia, either puerperal or renal. He believes that whatever may be the pathology of this condition, whether due to deficient urinary elimination, or the presence of too much lead in the blood, or to functional derangement of the liver, or some other gland, under the influence of a toxic agent, it is certain that when the attacks occur, the constitution of the blood is abnormal, and it contains noxious principles which should be eliminated. If we wait for the organism to relieve itself by the natural emunctories, the attacks continue and may terminate in a fatal manner. The most energetic purgations, diuretics and diaphoretics act too slowly, while the treatment suggested by Desplats is more prompt and sure. By the latter means elimination takes place rapidly, and the serum injected modifies the composition of the blood. He considers this method also applicable in puerperal or renal eclampsia, and in other affections where toxins play the principal rôle. FREEMAN

27. EPILEPSY; ITS SURGICAL TREATMENT. McGrew. (*Medicine*, May, '97.)

A man of 36, received, in 1882, an injury to the head, about which little could be learned except that he was not trephined. A half hour after the accident he had a convulsion, and from then to the time of the operation, except for one short period, he had at least one fit a day. In 1894 he was operated upon, with unsatisfactory result. When first seen by the author, later in that year, he was having numerous fits, preceded a few minutes by a peculiar headache as an aura. Examination showed, on the left side of his head, two scars, one linear, the other horseshoe-shaped, commencing about 2 cm. laterally from the junction of the posterior and middle thirds of the line joining the glabella and theinion, and passing forwards and downwards. These were sensitive on pressure. On March 28, 1895, the patient had a fit, and lost the power of speech and hearing, but could read and express his wishes in writing. This condition persisting, on Dec. 5, 1895, the author exposed the bone in the left parietal area by a large horseshoe flap. The dura was found adherent to the bone about the margins of a semilunar opening, 2 cm. long by 8 mm. wide at its widest part, left from the earlier operation. The opening was rounded and enlarged to the size of a silver dollar, the dural adhesions separated and clipped away, and the edges of the bone made smooth. The dura was not opened. The outer table of the skull was beveled, a thin silver plate shaped like a watch glass was fitted over the opening, and the flap closed over it. The patient made a good recovery. As soon as he came from under the anesthetic he could speak and hear, and during the fifteen months which have elapsed during the operation he has had no fits.

C. L. ALLEN.

Book Reviews.

NORMAL AND PATHOLOGICAL CIRCULATION OF THE CENTRAL NERVOUS SYSTEM. By Wm. Browning, Ph.B., M.D. J. B. Lippincott Co. Philadelphia, 1897.

Dr. William Browning's work on the normal and pathological circulation in the central nervous system is a welcome addition to our knowledge of a subject so difficult of investigation. The author fully recognizes these difficulties which lie in the fact that the conditions of the circulation by the very process of the experimental study are changed from those existing *intra vitam*.

The author scarcely gives sufficient importance to the comparatively free passages of the cerebrospinal fluid from the brain into the spinal cord, and vice versa, thus relieving within certain limits unequal pressure on the brain as a whole either in hyperæmia or anæmia.

There seems little doubt that this is an important factor in the altered cerebral pressure which must follow in cerebral hemorrhage, or softening, or in cerebral growths.

Many interesting and unusual cases of internal hydrocephalus and symmetrical central lesions are related in more or less detail. From the careful investigation of the literature double lesions are found to be more common than is usually supposed. The usual cause of cerebral hemorrhage or thrombosis is disease of the cerebral vessels which is general and not localized unless following some local injury. It is easily supposable that one part of this chain of diseased vessels might be more affected than another, thus leading, as is more frequently the case, to a unilateral, rather than a multiple or symmetrical lesion. The exciting cause of cerebral hemorrhage is said to be "nerve action, most probably vasomotor." The muscular coat of the cerebral arteries is subject to some influence leading to contraction and dilatation, but whether this is accomplished by the direct influence of the circulatory fluid altered, as it may be, in disease, or by nerves in the cerebral vessels, is still a question for further elucidation.

The author relates two cases of interest, in which hemorrhage probably followed a previous softening in some area. This condition is not always easy of proof. In these studies of Dr. Browning we have many suggestions of great importance.

E. D. FISHER.

TRAUMATIC INJURIES OF THE BRAIN AND ITS MEMBRANES. With a Special Study of Pistol Shot Wounds of the Head in their Medico-legal and Surgical Relations. By Charles Phelps, M.D., Surgeon to Bellevue and St. Vincent's Hospitals. 8vo, 582 pages. With 49 illustrations. Cloth, \$5. New York: D. Appleton & Company. 1897.

This contribution to regional surgery, neurology and legal medicine is based upon the study of five hundred intracranial traumatisms which were observed in the author's own hospital services and in those of his colleagues, and upon the results of experimental pistol shot wounds of the head made during the past three years in the city morgue. The work is composed of three parts. The first describes the pathology, symptomatology, diagnosis and treatment of general

brain lesions, and the second has to do with the surgical and medico-legal relations of pistol-shot wounds of the head. The last hundred and ninety pages are taken up by the histories of the clinical cases, two hundred and twenty-five of which are amplified by reports of the gross appearances found at autopsy. The whole is preceded by a consideration of fractures of the skull, and in this part are to be found valuable statistics as to the most frequent seats, with the attendant mortality, of the bone lesions, together with such symptoms as are characteristic of each class.

In the section on pathology objection is made to the continuance of the term "concussion of the brain," although the author proposes no satisfactory substitute to designate the losses of consciousness, incident to head injuries of moderate severity, from which the patient quickly recovers without ever presenting subsequent symptoms of a character to indicate any actual disturbances of brain structure. Considerable prominence is given to meningeal contusion as a cause of general cerebral symptoms. In it is found the explanation of the peculiar circumscribed sub-pial collections of clear fluid which seem to be the only discoverable causes of death in some cases. The author does not believe that this condition can be diagnosticated during life, since "when the edema is considerable in amount, it is still insufficient to occasion symptoms of compression." It may be said, however, that at the last meeting of the American Neurological Association, circumscribed edema was accepted as an occasional cause of focal symptoms (see this Journal, August, 1897).

The chapters on symptomatology contain many interesting observations as to the distinctive clinical features of the different kinds of brain lesions (classified as 1. Hemorrhages; 2. Diffuse and limited contusions; 3. Laceration; 4. Secondary inflammations), and as to such symptoms as result from affections of definite cerebral territories. The cases of injury to the frontal lobes are so noteworthy that they may be referred to in some detail. Twenty-eight cases, confirmed by autopsy, are considered as permitting an estimate of the direct results of frontal lesions. The laceration involved the left frontal lobe in eleven, the right in seven, and both lobes in ten of the cases. "In the eleven cases in which frontal laceration was confined to the left lobe, there was mental aberration or deficiency, apart from mere stupor or delirium, in every one; while in the seven in which laceration was confined to the right lobe, it was observed in none. In the ten cases in which frontal laceration involved both lobes . . . specific mental disturbance . . . was observed in eight." The two others were not clinically available for statistics. The importance of these results is self-evident. If subsequent and more extended observations confirm them, Dr. Phelps will have succeeded in pointing the way to the solution of knotty psychological problems. The discussion of the symptomatology of brain lesions is continued by the consideration of the diagnostic significance of paralysis, morbid movements, conjugate deviation of the head and eyes, the condition of the sphincters, pulse, temperature, etc. These are again referred to under "Diagnosis." The first part concludes with a chapter devoted to the principles of treatment.

The chief interest in Part II. attaches to the medico-legal relations of pistol shot wounds of the head. Dr. Phelps has long been interested in this subject, and the present systematic exposition of the results of his investigations is a valuable contribution to this branch of forensic medicine. He shows the inferences as to the size of the ball, the length of range, etc., which may be drawn by examination of the wound of entrance and exit, the burning of the skin and hair, the deposit of powder grains in the skin or in the track of the

bullet. This part of the book is fully illustrated with excellent photographs of the cases.

The work as a whole merits the serious attention of all who have to do with encephalitic traumatism. It embodies the results of studious analysis of a very large number of cases and consequently contains a fund of useful information. The neurologist, however, may find the histories deficient in points of detailed examination, and will regret that the specimens from some of the especially interesting cases were not examined by means of the microscope. The part of the book which treats of pistol shot wounds fills a hitherto conspicuous gap in our literature and will at once be accorded the place of authority on this class of injuries.

There are places in the text where the style might have been casier and more clear, and the book would have had a more convenient usefulness had it been indexed.

PEARCE BAILEY.

TWENTIETH CENTURY PRACTICE. Vol. xi. Diseases of the Nervous System. Edited by Thomas L. Stedman, M. D. William Wood & Company, New York, 1897.

An article by Lloyd, of more than four hundred pages, on Diseases of the Cerebrospinal and Sympathetic Nerves, is the first of a series of excellent papers. The author has drawn largely on the experience gained in an active practice, and on the best literature relating to the peripheral nerves. The Trophoneuroses are treated by Mills and Dercum. Bruns and Windscheid write on Diseases of the Spinal Cord; Möbius on Tabes Dorsalis; Strümpell on Combined System Diseases of the Spinal Cord; and Witmer, from the standpoint of the psychiatrist, on Pain. The volume will prove useful, not only to the neurologist, but to the general practitioner and the student.

BOOKS RECEIVED.

"An Epitome of the History of Medicine," by Roswell Park, M. D. F. A. Davis & Co.

"Die Darstellung krankhafter Geisteszustände in Shakespeare's Dramen." Von Dr. Hans Laehr. Paul Neff Verlag, Stuttgart.

"Index Catalogue of the Library of the Surgeon General's Office," U. S. Army, Vol. ii. B to By water. Washington, 1897.

"System Nerveux Central, photo gravures," Dr. J. Dagonet, Libraire. J. B. Baillierrie Fils, Paris, France.

"Die Akromegalie." Von Dr. Maximilian Sternberg.

"Sulla Opportunità ed Efficacia della Cura Chirurgica, Genecologica Nella Nevrosi Isterica E Nella Alunazione Mentale," Dr. G. Angelassi and Dr. A. Pieraccini.

"Dyskinesias Arsenicas," (Nova Contribuicas E Estado Actual de Questas.) Dr. Deslinde Galvas.

"Sitzungsberichte der kaiserlichen Akademie der Wissenschaften" 3 numbers.

"L Hysterie Aux xvii et xviii Siecles," Mme. G. Abricosoff. G. Steinheil, Paris.

"Iowa State Medical Society Transactions," Vol. xv.

"Die Bedeutung der Augenstörungen für die Diagnose der Hirn und Rückenmarkskrankheiten." Dr. Otto Schwartz. S. Krager. Berlin.

"Studies Gummous." (Tertian) Syphilis, by Kristian Grou. Steenske Bogtrykkerj, Kristiania.

THE
Journal
OF
Nervous and Mental Disease

Original Articles.

ON ARRESTED DEVELOPMENT AND LITTLE'S
DISEASE.¹

By WILLIAM G. SPILLER, M.D.,

With Remarks by W. W. Keen, M.D., On the Advisability of Operation in Microcephaly.

The diagnosis of arrested development is so frequently made that the desire naturally arises to know the facts obtained by actual examination of brain and cord in such cases, and in this paper an attempt is made to present a few of these, especially those relating to arrested development of the motor tracts. The clinical notes have in great part been taken from the casebook of Prof. Keen, at the Philadelphia Orthopedic Hospital and Infirmary for Nervous Diseases. I am deeply indebted to him for the liberality he has manifested in placing them at my disposal. Use has also been made of the paper published by Prof. Keen in the American Journal of the Medical Sciences, June, 1891. The case K. K. is from the Pennsylvania Training School for Feeble Minded Children.

The parents of the child K. K. were first cousins, and of moderate intelligence. One child, still living, has hydrocephalus; another died in his fourth month in convulsions.

¹ From the Wistar Institute of Anatomy and Biology. One of two papers presented for membership in the American Neurological Association.

K. K. had a very small head at birth, and the anterior fontanelle was closed. She was born in difficult labor, at full term, without the use of instruments, and was the second child in the family. The mother fell down ten steps and hurt her side in the seventh month of pregnancy. No history of venereal disease in the parents could be obtained. At the age of nineteen months, K. K. was unable to sit erect without support, though she could hold objects in her hands. She had moderate contracture of the flexor muscles of the feet, but her legs were not paralyzed. The knee-jerks were absent, possibly on account of contracture, and ankle-clonus was not observed. The child presented the appearance of an idiot, and her attention could only be obtained momentarily. Convulsions occurred in the teething period. The measurements at the age of nineteen months were as follows:

Biparietal	10.3 cm. (4 1-16 in.)
Biauricular	10.2 cm.
Bitemporal	8.3 cm. (3 1-2 in.)
Occipito-frontal	11.9 cm. (4 3-4 in.)
Circumference	36 cm. (14 1-4 in.)

By comparison with average measurements it will be seen that the head was not much larger than that of a fetus at full term. Craniotomy was performed when the patient was nineteen months old. The description by Dr. Keen of the operation is as follows: "An incision was made one inch to the left of the middle line, parallel to the sagittal suture, and six inches in length. A curved incision was then made from the anterior end of this line downward, so as to lift a frontal flap, the scar of which would be hidden by the hair. A half-inch button of bone was removed by the trephine, and from this anteriorly and posteriorly a furrow, a quarter of an inch wide, was cut out of the bone, extending to within an inch of the supraorbital ridge and an inch above and to the left of the inion. The length of the furrow was five inches. The amount of hemorrhage from the scalp was very slight, much less than I have

found in adults. The bone was very thin, about one to one and a-half mm. thick only, but bled freely. Opposite the parietal boss the dura was very adherent to the bone, but at all other points was separated easily. When the point of a pair of scissors was put under the flap of bone thus loosened, and the handle of the scissors let down gently, the simple weight lifted the flap perceptibly. The periosteum corresponding to the bone removed was cut away. The dura had not been opened, and appeared normal. A few strands of horsehair were placed in the furrow, and the wound dressed. The operation lasted half an hour. The temperature at its close was 98 deg."

Three months later a linear craniotomy was performed in precisely the same manner on the other side of the head.

The condition of the child was thought to be improved to some slight degree after the operation, though this improvement may have been due to the attention she received at the Children's Hospital from the chief nurse, Miss Hogan. When two and a half years old, K. K. could only say "Mammy." The arms and legs were freely moved, but incoördinately, and motion was greater in the former. She could sit up without support, but if placed upon her feet she fell at once to the floor. Her intelligence was of very low grade.

In Dr. Llewellyn's report of her condition during her residence at the Pennsylvania Training School for Feeble-Minded Children, and just before her death, the statements are made that K. K. could not stand; could not feed herself, and had little use of her hands, though she could understand many things said to her.

She died suddenly at the age of six. When seen after death the feet were in the position of marked talipes equino-varus, and the toes were flexed. The thenar and hypothenar eminences of the hands were of nearly normal development; the thumbs were not flexed; the first phalanges of all the fingers were extended, and the second and third moderately flexed. Her form was very slight. The

circumference of the head through the glabella and inion was sixteen inches. In removing the calvarium the dura was found to be very adherent near and along the sagittal suture. The brain was small, and weighed sixteen ounces avoirdupois. The pia was not adherent. There were no abnormal depressions, and no sclerotic areas, and the gyri and fissures were well developed. There was not the slightest indication of microgyria. The cord was very small.

From the clinical description, which has been confirmed by several, as the child was studied by a number of physicians, the affection was not unlike the disease described by Little, which in France and Germany is more frequently called by his name than in England. The labor was difficult, but neither this nor premature birth was the cause of the child's condition. The definition which Brissaud² gives of Little's disease, which is in reality merely a symptom-complex, is as follows: "Spastic and congenital paraplegia of the four limbs, more pronounced in the inferior, occurring especially in children born before full term, characterized by spasm more than by paralysis, and not associated with convulsions or disturbance of intellect, and capable of progressive amelioration, if not of complete cure." Little,³ however, spoke of impairment of the intellect. Freud⁴ says there are cases of diplegia in which premature birth and difficult labor are not the causes of the paralysis, but that there is a predisposition to paralysis in these children which, if sufficiently strong, may develop into diplegia, without the occurrence of trauma. The majority of children born before full term, or in a condition of asphyxia resulting from dystocia, do not present diplegia. This case of K. K. is of unusual interest, for Marie⁵ states that there has as

² Brissaud: *Maladies Nerveuses*, p. 110.

³ Little: *Transactions of the Obstetrical Society of London*, vol. iii., pp. 306, 307.

⁴ Freud: *Revue Neurologique*, 1893, p. 183.

⁵ Marie: *Traité de Médecine*, vol. vi., p. 457.

yet been no autopsy in a case of Little's disease.⁶ He thinks dystocia or disease of the mother or fetus in certain cases may possibly produce spastic dorsal tabes (Little's disease) in children born at term, but the most common cause is premature birth, and the condition is one of imperfect development of the pyramidal fibres, as is seen in the case K. K. Emphasis must be laid on the statement that *there was no macroscopic lesion* in my case. It is impossible to state the degree of spasticity which was manifested by the child K. K., inasmuch as I never saw her in life, but the contractures of the flexor muscles of the feet at the age of nineteen months and after death indicate that the muscular tonus was exaggerated. The child also was not paralyzed. To those who object to the title of Little's disease for this case on account of the birth at full term, the presence of microcephaly, and the insufficient degree of spasticity, the name of cerebral diplegia in the sense used by Freud,⁷ or of "infantile spastic state of cerebral origin (*états spasmodiques infantiles d'origine cérébrale*) suggested by Van Gehuchten,⁸ may be more acceptable.

Sections from the superior part of the ascending frontal convolution of the right and left sides of the brain of K. K., in the portions which contain the centres for the lower extremities, and especially from the paracentral lobule, show an absence of the very large ganglion cells, (*Riesenzellen*), which are found in a normal motor cortex. The cells of the third layer (Cajal) are a little larger than those of the second, and some are of very good size; but there is great diminution of the giant cells which are present in sections from a normal brain of a child of about the same age, taken from the same portions of the cortex for comparison. It may, however, be possible to find one or two cells of unusual size in a field, but they

⁶ The cases of Dejerine reported to the Société de Biologie were published after this paper was written.

⁷ Freud: Zur Kenntniss der cerebralen Diplegien des Kindesalters.

⁸ Van Gehuchten: Revue Neurologique, Feb 15, 1897.

are always much smaller than the largest of the giant cells of a normal cortex. The condition can hardly be attributed to the age of the child. Some of the few cells present in the anterior horns of the cord are large. The pyramidal cells of Betz⁹ are said to be the largest cells in the entire nervous system, excepting some in the spinal ganglia, and are found in the motor cortex. They resemble the cells of the spinal cord. Betz stated that they are found chiefly in the fourth of his five cortical layers; that they are less numerous in the lower half of the anterior central gyrus, and are most abundant in the paracentral lobule. He has found them in the brain of idiots (*Riesenpyramiden, Nervenriesenzellen*). V. Monakow¹⁰ cut the fibres of the inner capsule of a cat. The histological examination of the motor cortex was most astonishing, according to his statements. Betz's giant cells had entirely disappeared, while the other layers presented no noteworthy changes. Mahaim¹¹ says that the connection of the giant cells with the pyramidal fibres has also been demonstrated by v. Gudden and Moeli in the rabbit and guinea pig, and by Moeli and Henschen in man.

The apical processes of the pyramidal cells in the cerebral cortex of K. K. point toward the surface, and do not cross one another in the irregular arrangement described by some writers. The anterior pyramids of the oblongata are small, but present no evidences of degeneration. It has not been possible to obtain an oblongata or cord from a child of exactly the same age for comparison, but the whole central nervous system is much smaller than that from another child of seven years. There are very few cells in the anterior horns of the cervical swelling, though those which are present have a normal appearance. The horns are small. The crossed pyramidal tracts and columns of Goll are decidedly less intensely stained

⁹ Betz: *Centralblatt für die medicinischen Wissenschaften*, 1874 and 1881.

¹⁰ V. Monakow: *Neurologisches Centralblatt*, 1883.

¹¹ Mahaim: *Archiv für Psychiatrie*, xxv.

by the method of Weigert. In carmin sections an increase of the neuroglia is very evident in the columns of Goll, and while the fibres of the anterior and lateral columns are small, those in the crossed pyramidal tracts are of unusually fine calibre, and are smaller than those in Goll's columns. The thoracic portion of the cord is very small. The anterior horns of the lumbar region contain a few cells of normal appearance. The anterior and posterior spinal roots are normal. Serial sections have been made of the central nervous system from the oblongata into the thalamic region, but they have not revealed anything further worthy of special note. This case K. K. seems to be similar to the one described by Mya and Levi (quoted by Van Gehuchten). Their patient was born in difficult labor at full term, was of feeble intellect, and presented general muscular rigidity. Partial agenesis of the pyramidal tracts in the cord, and incomplete development of the pyramidal cells of the motor cortex were found.

Ganghofner¹² has reported a case of what was apparently general spasticity. Sections from the upper part of one anterior central gyrus appeared normal, but a scarcity of fibres in the crossed pyramidal tracts of the cord was noticed. Macroscopically the brain and cord were normal, and yet he believes that the cortex could not really have been perfectly developed. In two other cases of spasticity in which no peculiar findings were observed by the microscope he thinks there may have been a moderate decrease of the pyramidal fibres which eluded detection.

Otto¹³ has reported two cases of idiocy. One of his patients was unable to walk or stand, but could move the limbs feebly while in bed. He could neither speak nor understand anything said to him. The other child presented contractures in all the extremities. Microgyria was found in both cases at the autopsy, and in the microscopical investigation decided anomalies were observed in

¹² Ganghofner: *Zeitschrift für Heilkunde*, vol. xvii., 1896.

¹³ Otto: *Archiv für Psychiatrie*, vol. xxiii., p. 153.

the cellular elements, although the fibres of the cerebral cortex presented no great variation from the normal. The cells were quite numerous, well developed, and regularly placed in the layer of small pyramidal cells. The large pyramidal cells were almost entirely absent, and those that were present were little larger than the small pyramidal cells. There were no giant pyramidal cells at all in the frontal and central lobes, but there were many round ganglion cells. In his cases the pyramidal tracts in the oblongata contained fewer fibres than normal, but they were not degenerated, and this deficiency of fibres he attributes to the scarcity of cortical ganglion cells. The ganglion cells of the cord, contrary to the condition in my case, were well developed. In Anton's case the pyramidal tracts were very small, and the central gyri were affected as in Otto's case (Otto). Otto states that these giant cells of the cerebral cortex are developed in normal brains at the time of birth. He found also pale gray areas in the white matter of the hemispheres which were formed by collections of ganglion cells between the fibres of the white matter. They were evidently nerve cells, for many were pyramidal in shape and had processes.

Binswanger¹⁴ also has reported a case of microgyria. The child was unable to stand, walk, speak, or understand what was said to him. The limbs were contracted. The central gyri in both hemispheres were absent. In sections from the left frontal lobe near the defect the giant cells (*Ricsenzellen* of Betz), were absent. There was no secondary degeneration of the pyramidal tracts of the cord, and the cells of the anterior horns showed no pathological changes. The fibres in the white matter of the cord were possibly a little smaller. The anterior horns, especially in the lower part of the cord, were relatively small, and the cells seemed to be less numerous.

In the case of idiocy with microcephaly, reported by Popoff,¹⁵ the cortical nerve cells were unequally distrib-

¹⁴ Binswanger: *Virchow's Archiv*, 1882, p. 427.

¹⁵ Popoff: *Archiv für Psychiatrie*, xxv., 1893.

uted, and in some places had an irregular arrangement. The apical processes of the pyramidal cells in normal brains are parallel to one another; but in Popoff's case, they crossed at an acute angle. In some portions there was a decrease in the number of nerve cells, and most of the cells were small and occasionally their contour was abnormal. The cortical vessels were more or less thickened. In the frontal and paracentral lobes the giant cells were quite hard to find, and had not attained a large size. The left cerebral hemisphere was more flattened and smaller in its anterior portion than the right. During life the right upper limb was adducted, and the forearm, hand and fingers were flexed.

Köster¹⁶ in the brain of an idiot found thickening of the neuroglia, enlargement of the pericellular and perivascular spaces, decrease in nerve cells in certain parts, and, in some, deposits of pigment. In some shrivelled cells there was no nucleus. He also found the irregular arrangement of the pyramidal cells. Some of these were parallel to the surface of the cortex, lying on their side. Köster gives a brief review of some of the earlier investigations on idiotic brains.

Oppenheim¹⁷ observed an increased number of the small round cells of the cortex in microgyria; the pyramidal cells in places were entirely absent, or imperfectly formed and abnormally placed.

Friedmann¹⁸ describes a case of infantile spastic paraplegia associated with idiocy. One hand was contracted. The brain and spinal cord, with the exception of the posterior columns, were smaller than normal. The pyramidal cells of the cortex were reduced to about half the usual number, the pericellular spaces were enlarged, the small vessels were distended, and their walls had undergone hyaline degeneration. The number of the vessels was less

¹⁶ Köster: *Neurologisches Centralblatt*, 1889, No. 10.

¹⁷ Oppenheim: *Neurologisches Centralblatt*, 1895, p. 131.

¹⁸ Friedmann: *Deutsche Zeitschrift für Nervenheilkunde*, vol. iii.

than normal. Some of the cortical pyramidal cells were altered in shape. Diffuse areas of sclerosis were found all through both hemispheres. The mass of white matter was greatly diminished. The pyramidal tracts and all the white columns of the cord, except the posterior, were small. Friedmann is inclined to attribute the condition in this case primarily to the changes in the small vessels.

B. Sachs¹⁹ has reported a case which is not so well known in the foreign literature as it deserves to be. He claims that it is the first case of congenital spastic paraplegia examined microscopically. In this case the labor was difficult, and the child was asphyxiated when born. Convulsions were frequent in early infancy. Convergent strabismus was present. The deep reflexes were increased. The mental development was deficient, but the child was not absolutely idiotic. It was one year old at the time of death. Sachs found thickening of the pia, characterized by general cellular infiltration; its blood vessels showed marked cellular proliferation, and the pia was adherent to the cortex. In the cortex itself there were few, if any, normal pyramidal cells. In the outer layers, and particularly in what would correspond to Meynert's third and fourth layers, there was an enormous profusion of small glia cells. The blood vessels were in part normal, but many of them showed marked small cell proliferation of the walls. There was unquestionably a thickening of the neuroglia. The anatomical diagnosis was chronic meningo-encephalitis. Sachs considered the case to be due to a wide-spread effusion of blood between the pia and the cortex at the time of birth. The spinal cord revealed a most distinct degeneration of both lateral columns, secondary to the cortical lesion. This degeneration could be recognized in all parts of the motor tracts. There is no degeneration of the lateral tracts in the case K. K., but the condition is one of partial agenesis.

In his work on the nervous diseases of children.

¹⁹ Sachs: New York Medical Journal, 1891, vol. i.

Sachs²⁰ speaks again of this case and states that the upper extremities were less paralyzed, but somewhat rigid. What at first he supposed to be secondary degeneration of the pyramidal tracts he later concluded was a primary and congenital defect. In another case of arrested cerebral development published by him,²¹ the cortical ganglion cells were very imperfectly formed, and were not numerous. There was no trace of a previous encephalitic process, and no change in the blood vessels. He speaks again of this case in his paper on amaurotic family idiocy.²² In the brain from the sister of this patient similar findings were observed, and in the lower part of the cord (the upper portion was lost), the lateral columns presented a degeneration that "was very different in character and extent from an ordinary secondary degeneration.". Sachs regarded the condition of the pyramidal cells of the cortex as a sign of arrested development and not of inflammation. Kingdon²³ also noted changes in the cortical pyramidal cells in his case of amaurotic family idiocy, as well as "well-marked descending degeneration" in the cervical region of the cord.

Schiff²⁴ found that after removal of the sigmoid gyrus in a dog twelve days old, the pyramidal tract of the opposite side in the cervical region stained more deeply with the carmine than normally, when examined seventeen weeks after the operation. On the same side of the cord there was a small red patch in the pyramidal tract. He was much surprised to find that there was no true secondary degeneration, but instead of this the pyramidal tracts were filled with thin nerve fibres with very small axis cylinders. He later observed this diminution in the size of the pyramidal fibres in six cases, and in all these

²⁰ Sachs: *Nervous Diseases of Children*, p. 399.

²¹ Sachs: *Journal of Nervous and Mental Disease*, 1887.

²² Sachs: *New York Medical Journal*, May 30, 1896.

²³ Kingdon: *Transactions of the Ophthalmological Society of the United Kingdom*, vol. xii., 1892.

²⁴ Schiff: *Centralblatt für Physiologie*, 1893, p. 7.

the area occupied by the small fibres was not the same. In none of these cases did he find any true degeneration, even when the animal had lived five months after the operation. In one case the greater part of both lateral columns was involved, and in another small fibres were found in nearly all the white columns. Schiff says it might be imagined that true secondary degeneration in young animals does not occur. This idea is incorrect, for Löwenthal²⁵ has shown that this may be found in a kitten fourteen days after section of the posterior part of the lateral column. Schiff has obtained this result also in young dogs. However, six cases prove the frequency of simple atrophy of motor fibres after a cortical lesion in early life. These fine fibres are present in the case K. K., but we must not forget that in a child the nerve fibres are smaller than in an adult, and do not attain their full development for a long time, as Hösel²⁶ has stated. Still, making allowance for this fact, there is no doubt that the pyramidal fibres are of uniformly finer calibre than those of any other part of the cord, and *much* smaller than in the child of seven years. They probably represent a condition of agenesis.

It will be noticed in Otto's case (l. c.) that there was no degeneration of the fibres of the pyramidal tracts, but merely a diminution in the number of fibres. It cannot be said that there is a diminution in the number of fibres in the case K. K. They are very small and, therefore, many are crowded together in a given area. Oppenheim²⁷ has reported atrophy of the direct and crossed pyramidal tract from porencephaly. There was no true degeneration, although a slight increase of neuroglia, and possibly a degeneration of scattered fibres. There are many similar cases in the literature. Gierlich²⁸ examined a case in which

²⁵ Löwenthal: Recueil zoolog. Suisse, vol. iv., p. 111, quoted by Schiff.

²⁶ Hösel: Archiv für Psychiatrie, vol. xxiv., p. 480.

²⁷ Oppenheim: Neurologisches Centralblatt, 1895, p. 132.

²⁸ Gierlich: Archiv für Psychiatrie, vol. xxiii.

the motor tract had been affected within the internal capsule very early in childhood. The pyramidal tract was smaller, but the fibres were not of diminished calibre, although their number was less than in the corresponding tract of the opposite side. There were no evidences of degeneration. His explanation for the paucity of nerve fibres and absence of sclerosis is that either a downward growth of motor fibres from the cortex did not take place, or else reactive inflammation did not develop after destruction and absorption of the young fibres. Hervouet²⁹ also thought that there was a deficiency of development of the nerve fibres of the lateral columns, without sclerosis, in his case of idiocy. In Popoff's³⁰ case of idiocy the gyri of both hemispheres were thin, but the left hemisphere was smaller than the right in its anterior part. The lateral column and horns of the right side of the cord were smaller than normal, and above the motor decussation the left pyramidal tract was small. This diminution in size of the right horns and lateral column was due to imperfect development, for there was no sclerosis, and the nerve fibres had probably never developed as in a normal cord. It has been known for a long time that micromyelia may be associated with microcephaly (Aeby, Thiele, Flesch, Schattenberg: quoted by Popoff).

Anton³¹ has reported four cases of fetal or early cerebral disease which caused deficient development of the pyramidal tracts. In one of his cases there was absence of the pyramidal tracts, and yet the gray matter of the cord was well developed. He believed that in complete agenesis of these tracts the spinal gray matter suffers very little or not at all, and states that no writer has reported changes in the anterior horns in micromyelia. This view is not tenable. In Popoff's case the right anterior horn

²⁹ Hervouet: *Archives de Physiologie*, 1884.

³⁰ Popoff: *Archiv für Psychiatrie*, vol. xxv.

³¹ Anton: *Ueber angeborene Erkrankungen des Centralnervensystems*, 1890, quoted by Popoff.

in the cord was much smaller and there were proportionally fewer cells within it, although the cells present were neither sclerosed nor atrophied. One side of the cord, including the anterior horn, was smaller also in Hervouet's case (l. c.); although the cells of the anterior horns were normal. In this case the columns of Goll presented the appearance of ascending degeneration, as in my case, and yet there was no lesion to be detected which could have caused an ascending degeneration. Hervouet regarded this condition of the cord as one of arrested development. The case K. K. also is contrary to Anton's view. The anterior horns of the cervical swelling are under-size and contain very few nerve cells. In Muratoff's³² case of bilateral porencephaly there were atrophied cells in the anterior horns of the cervical cord, and in the thoracic and lumbar portions scattered atrophic cells were found. The pyramidal tracts were degenerated, and this probably explains the condition of the ganglion cells of the cord. An abnormal condition of these cells probably frequently occurs when the pyramidal fibres are imperfectly developed in the child. In Muratoff's case No. VI., (an idiotic, microcephalic child), there was imperfect development of the pyramidal fibres, total atrophy of the cortical cells of the central lobe, and absence of the tangential fibres. The motor cells of the anterior horns of the cord were atrophied; the cellular processes were indistinct; and the cells though small were present in normal numbers. Railton,³³ in a case of double spastic hemiplegia, found the pyramidal tracts normal, and in the motor area (cortex?) there was a diminution in the number of the large ganglion cells and some increase in neuroglia. Marchand³⁴ says the nervous elements are in normal arrangement, though lessened in number, in uncomplicated cases of microcephaly.

³² Muratoff: *Deutsche Zeitschrift für Nervenheilkunde*, vol. vii.

³³ Railton: *British Medical Journal*, 1892, vol. i., p. 441.

³⁴ Marchand: *Abstract in Centralblatt für allgemeine Pathologie und pathologische Anatomie*, 1892, p. 780.

Steinlechner-Gretschischnikoff³⁵ in two cases of microcephaly observed imperfect development of the cord, chiefly in the pyramidal tracts, anterior columns and columns of Goll, and, in one case, in the direct pyramidal tracts. In one of these cases there were fewer ganglion cells in the gray matter of the cord. The statement by this authoress that the normal development of the columns of Goll, as well as of the pyramidal tracts, is dependent upon the development of the cerebrum, is in accordance with the findings in the case K. K. The experiments of Ceni³⁶ (of Milan) in hemisection of the posterior columns with the remarkable resulting changes in the cortical cells of the sigmoid gyrus and occipital lobe, chiefly of the side opposite to the hemisection, as seen by the silver method, would seem to show a connection between the fibres of the posterior columns and the cortical cells. The number of the cells in the anterior horns in microcephaly is usually less than in normal cords, according to Steinlechner-Gretschischnikoff.

Van Gehuchten³⁷ has found that the pyramidal fibres, even the axis cylinders, are entirely absent in the cord of a normal fetus of seven months, but are present in and above the anterior pyramids. Spastic rigidity in children born before full term, according to him, is not due to the absence of the myelin sheaths, nor to deficiency of the pyramidal tracts in their entire length, but to an arrest of the downward growth of these fibres. Flechsig claimed that the pyramidal tracts develop from above downward, and Starr³⁸ has offered a forcible argument in favor of this view from the study of a most extraordinary case of microcephaly in which the cerebral hemispheres were absent, and the pyramidal tracts had not developed.

Hervouet (l. c.) has shown that the pyramidal tracts

³⁵ Steinlechner-Gretschischnikoff: *Archiv für Psychiatrie*, vol. xvii., 1886.

³⁶ Ceni, quoted by Marinesco: *Semaine Médicale*, No. 48, 1896.

³⁷ Van Gehuchten: *Journal de Neurologie*, 1896.

³⁸ Starr: *Journal of Nervous and Mental Disease*, 1884, p. 343.

are not fully developed until about the fourth year of extrauterine life. Perhaps here is the explanation of the fact that the tendency to bilateral cerebral paralysis seems to cease in early childhood. The extrauterine cerebral paralysis in childhood is usually unilateral (Higier).³⁹ Sachs⁴⁰ has seen no case of cerebral diplegia or paraplegia which had begun after the fourth year, and he says that the tendency to bilateral cerebral affections seems to cease early in life. If the pyramidal fibres are so imperfectly formed in the first few years of the extrauterine period, they are more exposed to injury, and especially in their distal ends. It is not impossible that the degeneration seen in combined systemic disease, as for example in Strümpell's⁴¹ case or in many others, may be explained by the direction of the growth of the fibres. The sensory fibres grow upward in the cord, in the same way that the motor fibres develop downward. In combined systemic disease the pyramidal tract is usually most degenerated in the lower regions of the cord, and the posterior columns in the upper portion, i. e., in the parts last formed. Erb⁴² has expressed the opinion, held now by many writers, that the most distant part of the neuron may be the first to degenerate. In K. K. the paresis was more evident in the lower limbs. Van Gehuchten's views possibly offer the best explanation for this condition, for the lumbar cord in K. K. seems possibly proportionally smaller than the cervical, as though many of the cervical fibres had not developed downward into the lumbar region.

In examining some patients at the New Jersey Training School for Feeble-Minded Children, during the year 1896, the following cases seemed of sufficient importance in connection with this subject to be briefly described:

³⁹ Higier: *Deutsche Zeitschrift für Nervenheilkunde*, vol. ix., p. 31.

⁴⁰ Sachs: *Volkman's Sammlung klin. Vorträge*, 46-47, 1892, p. 448.

⁴¹ Strümpell: *Archiv für Psychiatrie*, vol. xvii.

⁴² Erb: *Deutsche Zeitschrift für Nervenheilkunde*, vol. i., p. 240, and *Neurologisches Centralblatt*, 1883, p. 481.



A. S. (Little's disease).

The patient A. S. presents great similarity in his appearance to the case K. K., which has been examined microscopically. The circumference of the head measures 17 1-2 inches. His intellectual powers are very feeble, and he never speaks. The saliva dribbles constantly. The entire body is covered with a papular eruption. The hands and feet are always cold. He has no power of motion in the lower limbs, and makes no attempt to stand. When placed upon his feet he sinks at once to the ground. Both

legs are much flexed at the knees, and cannot be extended by passive movement. The feet are in the position of talipes equinus, and can only be extended and flexed to a very slight degree by passive movement. The resistance in the thighs to passive movement is considerable. The patellar reflex is increased, but owing to the rigidity of the limbs the full degree of exaggeration is not perceptible. Ankle clonus and Achilles tendon reflex cannot be obtained on account of rigidity. In striking one patellar tendon slight adduction is observed of the opposite thigh (crossed reflex). The limbs and trunk are well nourished. The fingers are held flexed upon the thumb, but can be extended passively. The patient can very awkwardly pick up a large object with either hand. The reflexes of the upper limbs (at wrist, elbow, and biceps tendon), are exaggerated, but are partially checked by the muscular spasticity. The patient has some power of motion in the upper limbs. Strabismus is not present. As the boy understands a few simple commands, the sense of hearing cannot be destroyed. His vision apparently is good. The pupils are equal. He cannot sit alone, and unless supported he falls upon the bed. The child was born at the seventh month, in ordinary labor. As a baby he was strong, but "two years after birth he simply stopped developing, without any apparent cause." The mother was only seventeen when this child was born. One sister of the patient is living and healthy. It is stated in the history that the boy has said some words. He has never had epilepsy. He was born May 23, 1887.

This is a case of Little's disease, and is due to the cause which certain neurologists consider the only one which should be recognized, i.e., premature birth, and could his nervous system be examined lesions similar to those in K. K. might be found.

There are undoubtedly cases of arrested cerebral development which assume a paraplegic type. Sachs gave

the proof of this in 1891. I have found three feeble-minded children at the New Jersey Training School in whom the lower limbs were alone affected. One is selected as an example.

R. C., eight years old, is a paraparetic idiot. He has never spoken, does not understand what is said to him, and takes no notice of anything. The limbs and trunk are well nourished. He cannot walk, and makes no attempt to take a step. In the erect position he inclines backward, and has to be supported or would fall. He can move his legs when in the sitting posture. Knee-jerk and ankle-jerk are exaggerated, and slight ankle clonus is present. Above the hips the movements are good. The reflexes in the upper extremities are not notably exaggerated. Strabismus is not observed, and the pupils are equal. The head is well developed. This child was born at full term in easy labor. A few months after birth he had convulsions. As a babe he was said to be strong. His condition was congenital. Other children in the family are healthy.

Three or four months after the above description was written the patient died in status epilepticus, and fortunately a necropsy was obtained, and microscopic examination of brain and cord was made. Within a few days previous to death the patient was said to have had many convulsions. The body was much emaciated. The dura was somewhat adherent to the calvarium, and the pia, especially over the left parieto-occipital lobe, was congested. I am indebted to Dr. C. W. Burr for the material.

The brain, especially in the left hemisphere, presents a low type of fissuration. In the left hemisphere the parallel fissure unites with the Sylvian, the ascending frontal is almost fully united with the Sylvian, the first frontal is so distinct that it forms almost a straight line, and the Sylvian fissure extends almost to the upper border of the hemisphere.

In sections from the superior part of the right ascend-

ing frontal convolution, there is a diminution in the number of the pyramidal cells, and all are much smaller than the giant cells (*Riesenzellen*) of the normal cortex used for comparison. The nerve cells also present an appearance of imperfect development, though some are of fair size. In the paracentral lobule a few giant cells are present, but these cells are not nearly as numerous as in a normal cortex.

Within the cord all parts seem to stain equally well, and it is impossible to note any diminution in the number of the fibres in the crossed pyramidal tracts, and these tracts do not contain the many fine fibres observed in the case K. K. In this respect the contrast is striking. The cells of the cervical enlargement are numerous and well formed. In the lumbar region the motor cells are also large and abundant, and if there is diminution in the number of these it cannot be excessive.

How shall we explain the paraplegia? It is probable, as Ganghofner suggests in regard to his cases, that the fibres of the pyramidal tracts are really fewer in number, although the microscope is too poor an instrument to detect this. Everyone who has studied the nervous system will acknowledge that it is difficult, or even impossible, to detect a moderate decrease in the number of fibres in a given tract. The condition of the cortex justifies the opinion advanced. It is possible that proportionally more motor fibres in growing downward reached the cervical region than the lumbar.

There are other cases of undoubted arrested development in which the power of motion is not greatly affected. There is a picture of the following case in Mills' paper in Starr's⁴³ book on the diseases of children.

J. M. is a microcephalic idiot of low grade, and not taller than a child of four or five years. He was born January 31st, 1870. The circumference of the head is 16 1-4 inches. He can talk, but his words convey no

⁴³ An American Text-Book of the Diseases of Children. Edited by Louis Starr, M.D.

meaning. He has epilepsy, and may have as many as two or three attacks in a night, though on an average he has two or three in a week. He is very bow-legged, and this condition is evidently due to rachitis. He scratches and bites, and it is impossible to attract his attention. He does not understand anything said to him. He can walk, usually slowly, although he can be made to run. The right foot is planted firmly on the ground, but the left is kept elevated. His gait is not spastic. He moves the upper extremities freely, but slowly. The color of the irides is different; in one eye it is gray, in the other hazel. The right pupil is slightly larger than the left, and is irregular. Movement of the eyeballs is free. Sensation for pain is well preserved. The knee-jerk and ankle-jerk are exaggerated, and there is slight ankle clonus. Strabismus is not present. The reflexes in the upper extremities at the wrist, olecranon, and biceps tendon are exaggerated on both sides, but not so much as those in the lower extremities. One might be surprised to find motion in the extremities so good in a case of microcephaly of high grade, but paralysis is in no way due to small size of the head. Puberty has been attained. It is difficult to test the reaction of the pupils to light and accommodation, as it is impossible to make the man (?) keep his eyes open and fix an object. He was the third in the family, and was born at full term in normal labor. The father kept a saloon, and was inclined to indulge in alcoholic beverages. The patient had four sisters living and healthy in 1888. Six children have died from infectious diseases, and one from marasmus. There were no other malformed children.

After this paper was written my attention was called by Dr. Adolf Meyer, of the Worcester Lunatic Hospital, to the work of Hammarberg⁴⁴ which is of so great importance that a brief abstract must be given. The chief aim of this paper has been the investigation of the causes of

⁴⁴ Carl Hammarberg: Studien über Klinik und Pathologie der Idiotie, etc.

paresis and paralysis in cases of idiocy and microcephaly, and considerable evidence of the important part the giant cells (*Riesenzellen*) of the motor cortex have in the function of motion has been obtained. Hammarberg's work is confirmative of this view, and the abstract relates chiefly to his investigations on the ascending frontal gyrus. The results of his examination of the cerebral cortex must be read in the original. No abstract can do justice to a work of this magnitude.

While the chief aim of this paper has been the investigation of the causes of paralysis, the causes of imbecility have also in part been mentioned, for it must follow, that if the cortical zones which form the projection centres are arrested in their development during the embryologic period, or even the first month of extrauterine life, that this arrest will cause a corresponding arrest in the development of the association centres, as an inevitable consequence, and in this way an arrest in the development of the intellectual faculties (Flechsig, as quoted by Van Gehuchten.)⁴⁵

The giant-cells (Hammarberg) measure 35-40x50x80 microns in the anterior central gyrus. These have been called "gânglionic cells" by Bevan Lewis and Clarke.

Under the title of *Blödsinnige* (dements) four cases are reported by Hammarberg. The first child was twenty-two months old. During the first year of life it made no spontaneous movements. In the medial and upper half of the lateral aspect of the anterior central gyrus, as well as in the adjoining part of the frontal convolutions, the cells were arranged in one layer. They resembled the undeveloped cells of a fetus of five months, although they were larger than these, and yet smaller than normal cells, and they represented about one-fifth the normal number. A few undeveloped spindle cells were noticed.

Case 2. 14 years old. The child could not walk or sit erect, and there was paresis in the upper, and paralysis in the lower extremities. The anterior central gyrus.

⁴⁵ Van Gehuchten: *Journal de Neurologie*, January 5, 1897.

the posterior half of the lateral aspect of the inferior frontal gyrus, and the superior and inferior parietal gyri were the least developed. The cells were arranged in a single layer, and resembled embryonal cells, and were smaller than in normal persons of the same age.

Case 3. 10 years old. The cells in the anterior central gyrus were somewhat smaller and fewer than in normal brains. Groups of giant cells in normal number were observed. This case differed from the other two in that mobility was greater.

Case 4. 3 years old. The child could sit alone and walk, very insecurely, on level ground. In the right anterior central gyrus the pyramidal cells did not amount to one-fifth the usual number, and they were mingled with undeveloped cells. There were also cells which presented a hyaline appearance. In the lower part of the gyrus more large cells were noticed.

The larger part of the cerebral cortex in these four cases represented the normal degree of development in the latter half of fetal life. The different clinical symptoms could be explained by the cortical findings.

Two feeble-minded children of low grade are described:

Case 5. 26 years old. Movements were slow, but the patient could walk, although was soon exhausted. In the anterior central gyrus the cells and layers were somewhat smaller than in a normal person of the same age. The giant cells measured 25-30x50 microns. The slow movements could not be explained by the findings in the motor region. Possibly the decrease in the cells of the vermis was connected with the slow movements.

Case 6. 1 year, 10 months old. The child could not walk or sit erect. The imperfect mobility was not only due to the undeveloped condition of the cortex in the motor region, as this corresponded merely to the degree of development seen at the end of the first year, but also to the fact that the cells, and especially the giant cells, were much fewer than normal.

In these two cases the greater part of the cerebral cortex corresponded to the degree of normal development in the first years of life.

Three cases of moderate and slight grades of mental development are described:

Case 7. 14 years old. There was complete paralysis of the lower extremities and paresis of the upper. This was explained by the fact that the corresponding parts of the motor cortex were not more developed than in the first year of normal life, and the giant cells in the upper part of the anterior central gyrus were absent. In the lower parts of the gyrus the cells were larger and the appearance more like the normal.

Case 8. 12 years old. Mobility was not impaired. In the anterior central gyrus the cells were normal, although fewer. The giant cells were relatively most numerous.

Case 9. 16 years old. Both lower extremities were paretic, and the patient could not stand. The movements in the upper limbs and trunk were normal. The medial and upper sixth of the lateral aspect of the anterior central gyrus, the posterior central gyrus, and the inferior frontal gyrus were most altered, and the cells presented an undeveloped appearance. In the rest of the cortex the cells were normal, but fewer than in normal brains. There were no giant cells in the upper part of the anterior central gyrus.

In these three cases the number of the nerve cells in the greater part of the cerebral cortex was much less than the normal, and in a small area they represented the degree of development in the first years of life.

From the results of these important examinations Hammarberg concluded that the mental condition depends on the degree of development of the brain as represented by the cortical cells.

Surely in view of the testimony which has been presented by macroscopical and microscopical examination of the central nervous system in cases of microcephaly,

one must acknowledge that the operation of craniotomy is one which deserves most careful consideration. Perls and Edinger⁴⁶ have shown that hydrocephalus of mild degree occurring in childhood and not progressing, or rachitis involving the skull, have seemed to favor the mental development by lessening the resistance to the growth of the brain, but in these cases nerve cells were already present. It may be, as Starr⁴⁷ suggests, that operation stimulates the growth of cerebral tissue, but probably it can never cause the production of new nerve cells. We can never be perfectly sure of the nature of the lesion in any case of microcephaly. Dr. Keen has most kindly consented to give his views on the advisability of the operation of craniotomy in microcephaly, and the results obtained from his experience of many years are of inestimable value. I thank him most heartily for this important addition to my paper.⁴⁸

REMARKS BY W. W. KEEN, M.D., ON THE ADVISABILITY
OF OPERATION IN MICROCEPHALY.

I am glad that Dr. Spiller has been so fortunate as to be able to make a very careful post-mortem examination of the little girl K. K. reported in this paper. I cheerfully consent, at his request, to give my views on the present status of the operation of linear craniotomy.

My views are based not only on what I have learned from the experience of others, but upon a personal experience covering eighteen cases. Of these eighteen cases five died, a mortality of 21.7 per cent. The youngest was eighteen months old, the oldest six and a half years. As to their later history I can report of the thirteen who

⁴⁶ Edinger: *Vorlesungen über den Bau der nervösen Centralorgane*. Fifth edition.

⁴⁷ Starr: *Medical Record*, 1892, vol. i.

⁴⁸ Since this paper was written important works bearing on the subject of microcephaly and idiocy have been published by Pfleger and Pilcz (*Arbeiten aus dem Institut für Anatomie und Physiologie*, Obersteiner, No. v.) and by Kaes (*Monatsschrift für Psychiatrie und Neurologie*, No. i., 1897.)

recovered that six were slightly improved, seven were not benefited.

On the whole the prospect for improvement after the operation of linear craniotomy for microcephalus is not bright. Sometime since a patient called upon me who had sought advice as to her child, and when I told her that nothing could be done, the boy being simply an idiot with an average sized head and not the slightest indication favorable to operation, she was astonished to the last degree, and more than that she was heart-broken. She had been encouraged to believe that even in hundreds of operations not a death had taken place, and she stated that the very words used to her were that if she would allow an operation to be done "the result would be a revelation to her." Two specific instances of great improvement were cited to her. When I investigated these two cases I found that both were again in an asylum in a worse condition than before the operation. One can imagine what a crushing blow it was for a fond mother, who had anticipated a speedy cure for her idiot boy and a restoration to a normal intellectual life, to be told that such statements were not borne out by facts, and that in my opinion absolutely no good could result from operation.

I do not consider that a child with an average sized head is a suitable case for operation, nor secondly that in cases older, we will say, than about seven years of age, improvement can be expected. Nor do I regard the operation justifiable in those suffering from extreme microcephaly in which the head is *excessively* small. Only the cases showing a moderate degree of microcephaly are suitable for operation. The cases with *very* small heads, as the child K. K. reported in this paper by Dr. Spiller, as he has shown, have a very imperfect development of the nerve cells, which are essential for a normal or even approaching a normal life. In less extreme cases where probably the cells are more perfect we may hope for more. As I have said elsewhere (Medical News, November 29th,

1890, p. 558) "The inherent cause of microcephalus we do not know. Formerly it was supposed to be due to premature ossification of the cranial sutures, but the examination of several such skulls has shown that while this may sometimes be the case, yet in the cases examined there was no abnormality in the bony development of the cranium. On the other hand, we know that the growth of the skull keeps pace with the growth of the brain within it; and if the growing power of the brain be weak, a slight resistance on the part of its osseous envelope may be sufficient to check and stunt it."

The benefit so far as my observation goes is but slight. When the American Surgical Association met in Buffalo about three years ago, Dr. Roswell Park showed a boy, I think about twelve years of age, who had been very greatly benefited by the operation, so that he had, one might say, three-quarters of a normal intellectual development. I have never seen in any of my own cases any such improvement, and this case surprised me the more in view of the boy's age, which I think was about ten when he was operated on. Any real improvement, however, would justify the operation in my opinion. One of my cases for instance, was the child of a poor washer-woman with several other children. He was as restless, and as mischievous as a monkey. At night he was almost constantly crying, and so disturbed his mother that she had scarcely had one good night's rest since he was born. The operation transformed him into a quiet, fairly sleepful child, and was well worth any risk which attended the operation.

In fact, if any one may speak of risk, we can honestly only wish that in all the cases which should not be benefited the risk would be far greater than it is, since if such children cannot be helped it is far better for them, their parents and other caretakers and their companions that they should die.

I suppose I have declined to operate on at least one hundred cases which in my opinion were entirely unsuitable for operation.

My present conclusions, therefore, are:

(1) That in a moderate number of selected cases of medium degrees of microcephaly the operation will happily be followed by death in about fifteen to twenty per cent. of the patients.

(2) In a small number of cases benefit to a slight extent will be observed, but in the majority no results, good or bad, will follow the operation.

Were we to judge from the microscopic results in the case Dr. Spiller has so ably investigated, we should unquestionably say that no good result could ever follow, but we must remember that this child had an extremely small head, and was a case in which, with a now larger experience, I should absolutely decline to operate, and also, that this child did actually improve to some extent, whether as the result of the operation, or of the later excellent training and care that she had, we cannot judge positively. Clinically there is no question about a moderate improvement in a small number of cases.

After operation the greatest attention should be paid to developing the child's faculties by education. This can hardly be done in, or by any family. It is best done in a school such as that of the Misses Bancroft and Cox in Haddonfield, N. J., where a long familiarity with such cases enables them to adopt means to ends, to develop better methods of education which result from a wide experience, and to provide an individual teaching which neither in a family nor in a large school is practicable. How much improvement comes from the education and how much from the operation is often a matter of great uncertainty.

In all cases a clear statement of the mortality should be made to the parents and they should be prepared for a very probable disappointment in seeing only very slight good results or none at all, and then they must decide whether they are willing to accept the risk of death and the risk of disappointment.

GLIOMA OF THE RIGHT FRONTAL LOBE OF THE BRAIN.

By WILLIAM C. KRAUSS, M.D.,

Buffalo, N. Y.

H. S.—Single, aet. thirty-one; height, five feet eight inches; weight, one hundred and fifty-five pounds; occupation, architect and carpenter; complexion, dark.

Family History: Grandparents lived to old age. Parents are living and healthy at the age of fifty-six. There is no history of consumption or syphilis obtainable. One aunt on the mother's side died of cancer.

Early History: He passed through the usual diseases of childhood without any sequelae; grew rapidly and became an expert workman at his trade.

Present History: About a year and a half ago he noticed, while stooping over at his work, a little dizziness which soon passed off. Occasionally he would notice the vertigo when in an upright position, but on account of its transitory character he paid little attention to it. These dizzy spells continued without increasing any in intensity, and without further disturbances until February, 1896, when he began to have dull headaches with periods of exacerbation. The dull pain seemed to encompass the whole head, but during the exacerbation the pain became more acute and intense, and extended from the forehead to the nape of the neck; afterwards it became more intense and more fixed at the occiput, and was more or less continuous, so that he was obliged to discontinue his work (Sept. 20th, 1896.) When the pains were at their acme of intensity he would feel his head drawn backwards.

In May, 1896, while he and his brother were bicycling, they ran into each other, and he was thrown violently to

the pavement and received quite an extensive injury over the *left* eye, necessitating several stitches to close the wound. This accident did not seem to affect the headaches to any extent, but to another brother (a physician) seemed the probable cause of the head pains. It may be stated here that the patient was a rather quiet, uncommunicative man, especially in regard to his own feelings and sensations, and up to the time of the injury hardly ever spoke of his head pains. During the following three months he had but five severe spells of head pains.

About September 1st, 1896, he began to have spells of nausea and vomiting, generally in the morning when the stomach was entirely empty. These occurred at first about once a month, but increased in frequency until they occurred almost every other day. The vomited matter always consisted of a watery fluid, which he says had a very pleasant taste. The vomiting came on with a sort of hiccough and never prostrated him. During the summer (1896) the dizziness became more severe, and instead of having a dizzy spell about once a week he would feel dizzy every time he laid down or arose. When he tried to walk after having had one of these spells he staggered for two or three steps, but did not swerve to either side, never fell down, and noticed no other peculiarity about his locomotion. The eyes, he said, felt as if red hot irons were being thrust into them, also at times it seemed as if a white mist were hanging before the eyes; this would clear up and vision would become again good. He stated that his bowels were nearly always regular; that his appetite was variable; that he slept pretty well of late, though formerly he was disturbed by the head pains; that there was no difficulty in urination, no numbness or any other abnormal sensation, and that if it were not for the headaches and dizziness he would be able to work as hard as ever.

Status Praesens. Sept. 26th, 1896.

Mental condition: Conversation with him shows that

the mental faculties are intact, although he talks very little on any subject unless specially interrogated. He is in good humor, answers all questions promptly, and does not seem to take his sickness and enforced idleness much to heart. He reads the papers, does copying and makes himself useful about his brother's pharmacy. His actions and demeanor here show no change from his normal healthy condition, so his brother informs me.

His face is emaciated; the face lines are prominent; the complexion is dark, dull and ashen; the expression is somewhat apathetic. A very faint scar is seen over the left eye, the result of the bicycle accident in May, 1896; the scalp reveals no scars, no tender spots and no prominence of the sutures.

Percussion of the skull reveals no tenderness.

Eyes: The pupils are widely dilated, with no apparent difference between the two, and react to light and accommodation. The muscles of the orbits functionate normally in all directions. Ophthalmoscopically, a double optic neuritis is seen, and is, perhaps, a little more pronounced in the right disc. Hemorrhages have taken place about the fundus. Vision is subnormal, with no history of double vision.

The tongue and uvula show no deviation. No disturbance of audition, olfaction or gustation is found. The tendon reflexes, as well as the muscular, are normal. Sensation of the face, body and extremities is unimpaired. Dynamometric test gives the following results: Right hand, 140; left hand, 145; right leg, 182; left leg, 175.

Pulse shows eighty to eighty-five beats per minute and is regular and uniform. Urine contains no traces of sugar or albumin.

The persistent headaches, with dizziness, nausea, vomiting, and above all, choked discs, suggested a cerebral growth of some kind situated in some "silent" portion of the brain or else so small as not to call forth any localizing symptoms. Although he strenuously denied having

had syphilis, it was deemed prudent to put him on mercury and iodide, and to watch the effect of treatment, and to be prepared for the appearance of focal symptoms, so that if advisable surgical measures could be undertaken.

I saw him repeatedly after this, and apparently a change for the better was going on, as he complained less of his head pains; the vomiting and dizziness seemed to have lessened in frequency and severity, and he appeared more active and interested in his environment. This improvement was also observed by his brother, the physician, and the mercury and iodide were given with renewed vigor.

On October 17th, 1896, he received a letter from his former employer asking him if he were able to work again, and if so to write immediately. This pleased him greatly, and he could not understand why he was not able to resume work, inasmuch as he was feeling so much better. The optic neuritis, however, remained stationary, and was regarded more as an index of the condition in the brain than the subjective symptoms.

Between Oct. 17th and Dec. 8th, he thought he was so much improved that he refused to take any more medicine and did not come to see me. He also absented himself from his brother's office, and it was with some difficulty that he was persuaded to come this day.

On Dec. 8th, 1896, he reported as follows: He said his headaches had nearly disappeared; that he had not vomited in two weeks; that the dizziness was also disappearing; that the appetite was good and the bowels regular; that he slept well and "felt more like getting around." The papillitis of the left eye was receding, so that the outlines of the nerve were distinguishable. The right eye showed small hemorrhages about the optic disc which was much swollen. Vision of right eye was 20/30, left eye 15/30. Dynamometric test: Right hand, 145; left hand, 145; right leg, 180; left leg, 175.

Sensation, audition, gustation and olfaction were not

disturbed. Pulse was eighty, regular and uniform. His mind was clear, although he acted as if somewhat depressed and seemed to fall into a state of mental hebetude when no questions were asked him.

Believing him somewhat improved, it was decided to continue the anti-syphilitics up to the point of intoxication, especially as no localizing symptoms had appeared. It seemed from the subjective symptoms, the examination of the eyes and strength of the extremities that the growth was, perhaps, diminishing in size.

Soon thereafter reports came to me through his father and brother that the pains were becoming more intense, and were somewhat more paroxysmal, and that during these attacks he would say nothing, and even refused to answer questions, and was in a dazed condition. Pulse during a paroxysm ranged from forty-five to fifty-five.

On December 25th, he took dinner with his brother's family and seemed to be in the best of health and spirits. He played with the children, laughed, talked freely and enjoyed himself very much. The next day, Dec. 26th, he came to my office and denied having had any severe attack, and said the pains were in no way comparable to the pains he formerly endured. He also reported feeling pretty well, although his expression was not as cheerful as on former visits. Dynamometric test showed some diminution of power of the left hand. It was as follows: Right hand, 145; left hand, 135.

From this time on I received word from the brother that the paroxysms of pain were becoming so intense that he would be obliged to go to bed, and that in his opinion the patient was failing rather than improving. After one of these paroxysms (Feb. 1st, 1897), he urinated in the soap dish and into the bureau drawer, and was unable to find his way down stairs. This was the only time when the patient appeared at all irrational, and he recovered from this condition in twenty to thirty minutes. During

the intervals he would stay at the pharmacy writing labels, reading, and doing light work, and was possessed of all his mental faculties.

On February 4th, 1897, accompanied by his brother, he appeared at my office, and on first glance it was apparent that a change for the worse had taken place. The face was pale, ashen, dull and expressionless, the pupils of the eyes were widely dilated and his strength was evidently declining. He remembered nothing about the urination episode, nor of the several paroxysms, but knew about everything that had transpired during the time when his head was free from pain. He answered all questions intelligently, but, as on a former visit, lapsed into a dreamy, listless condition when left alone.

I was firmly resolved to locate the growth to-day if possible, and made a thorough examination.

Again the head was not sensitive to percussion; the eyes were in practically the same condition as before; audition, olfaction, gustation, and the various forms of sensation were likewise unaltered. Dynamometric test resulted as follows: Right hand, 135; left hand, 120; right leg, 170; left leg, 165.

There was no history of spasms or of periods of paresis of the extremities, while the face, tongue and uvula functionated normally. The pulse was eighty, regular and uniform. I informed the brother that in all probability the growth was not located in the parietal, temporal or occipital lobes, nor at the base, and was not affecting the basal ganglia, and that only by exclusion could it be located in one of the frontal lobes. And yet, even if here, it had not so far called forth any of the mental disturbances often met with in disease of the frontal area. Whether it was on the right or left side was also difficult to determine, and if on the left side, certainly did not affect the caudal part of the third, or of the second convolution near the Rolandic area. It did not affect the centres of

motor speech and writing, as neither aphasia nor agraphia was present. If the tumor were located on the right side of the brain it might have invaded certain areas whose functions are as yet undetermined, these "silent" areas being more extensive on the right side than on the left, and causing no definite symptoms when destroyed, as far as we know; or the cortex at the base of the brain, as that of the orbital convolutions, might have been diseased, and no recognizable symptoms have been present. This was, however, mere speculation, and the frontal lobes were thought to be the seat of the growth more than any of the other areas whose functions are unknown. The patient had several very severe attacks of head pain during the next few days and an unusually severe paroxysm on Feb. 10th, 1897, necessitating heroic doses of morphia. Following this attack he declined rapidly and died on the same day, Feb. 10th, 1897, at ten o'clock, P. M.

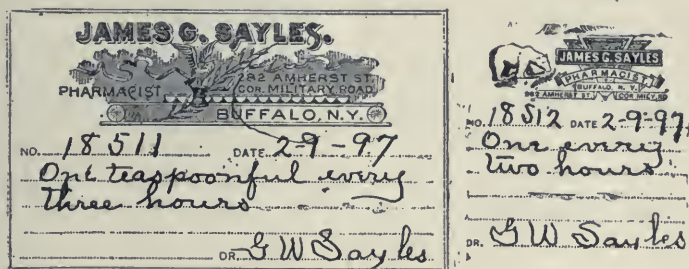


FIG. I.

On Feb. 9th he was at the pharmacy copying labels, a sample of which is here presented. He had not changed any since Feb. 4th, either physically or mentally, and no new symptoms had presented themselves. During the day he played whist, had on the boxing gloves, read, laughed, and felt better than for a month past.

The autopsy was made by Dr. Sayles and myself on Feb. 11th, 1897. The frontal bone over the left eye showed no traces of any injury which the bicycle accident

might have inflicted. The calvarium was of the usual thickness, and not adherent to the dura. The dura was not injected, and over the right frontal lobe fluctuated and showed an area of depression as compared with that of the left side. It was soft superficially to the touch, but dense and hard under pressure. The left lobe felt firm and resistant, and it was evident that the right lobe was the seat of disease. On removing the dura a large cyst, about the size of a walnut, the ectal wall of which was evidently the pia, was found in the right lobe at the surface. The cystic contents were collected in a bottle, and the collapsed walls brought into view a large, dense, firm tumor. The brain was removed and placed in a five per cent. solution of formalin for hardening, so as to facilitate a careful anatomical and pathological examination of the affected lobe.



FIG. II

The tumor occupied the middle and caudal portion of the first, second and third frontal convolutions, extending as far caudad as the ascending frontal convolution, thus occupying about one-half of the convex surface of the frontal lobe. Entad, the tumor extended to the lateral ventricle.

This cystic opening communicated with the lateral ventricle. Histologically the growth appeared to be a glioma with considerable connective tissue stroma present.

While the brain was in process of hardening an area of softening was felt over the angular convolution of the *left* lobe, and thinking that the formalin had not infiltrated the brain thoroughly, and that this portion was beginning to soften and break down, I cut into this region and found a cystic cavity about the size of a pigeon's egg. The fluid was similar to that which had escaped from the cyst in connection with the glioma. The cystic fluid from the tumor, which was about two ounces, was of a pale straw color, rich in albumin, and under the microscope revealed no traces of echinococci.

Two important questions arise in the study of this case: First, were the symptoms sufficient to warrant a positive focal diagnosis, and secondly, was it a mistake to postpone and eventually deprive the patient of the benefits of an operation.

It is now almost universally conceded that the right frontal and right temporal lobes contain no distinctive focal centres, and they are known as the "silent" or "latent" lobes or areas, in contradistinction to the corresponding lobes on the left side, which are the centres of motor speech and writing, and of hearing. The frontal lobes, perhaps equally, except for motor speech and writing, are supposed to be the seat of the intelligence and the psychical attributes which characterize mankind. Experiments on animals and clinical study have thus far failed to show just how much responsibility each lobe assumes in preserving the proper mental balance of the individual, and when the posterior portion of the second and third frontal convolutions of the left side are not involved, it is almost impossible to say, from the subjective symptoms, which lobe is the seat of disease. When along with marked mental decadence, aphasia or agraphia is present with the symptoms denoting brain tumor, then it is safe to localize the disease at least in the left frontal area, but still unsafe to say whether or not the right frontal area is also involved.

Inasmuch as the left side of the brain, especially the frontal and temporal lobes, contain important centres not present on the right side, may not the centres presiding over reason, memory, intelligence, or the higher psychical states be more localized in the left frontal lobe close to the centres of speech and writing, which are the two great channels through which the intelligence of the being is made manifest to the external world? The close proximity of the centres of speech and the centres presiding over those organs (articulatory) by means of which speech is possible; also the proximity of the centres of writing and those presiding over the movements of the fingers by which writing is possible, might suggest a like proximity of the centres of intelligence, and those centres through which the intelligence of the individual is expressed, namely, of speech and writing; all these are located in the left frontal lobe of the brain.

The symptoms presented by my patient would certainly tend to corroborate this view, and also to prove quite conclusively that the writing centre is not situated in the right frontal region. In my opinion, the areas presiding over the functions just mentioned enjoy much closer communion than we have as yet pretended to grant them. The same is true of the centres presiding over motion and sensation. In the motor areas, for instance, the layer of large cortical ganglion cells is interested in the movements of the various extremities, while the layer of small ganglion cells in the same cortical area could very properly preside over sensation. This view is not so very extraordinary, inasmuch as cases are on record of paralysis of motion and sensation where the lesion was found wholly in the motor areas. In like manner the centres of intelligence, inhibition and judgment might be located in the smaller ganglion cells of the whole frontal lobe of the left hemisphere, the centres of speech and writing occupying localized areas in the deeper layer of ganglion cells.

In fifty cases of tumor of the prefrontal area analyzed by Williamson,¹ the mental symptoms were generally well marked, and in many cases were the most prominent and earliest symptoms. His results are as follows:

There was a condition of mental decadence with a dull mental state, a loss of power of attention, a loss of memory, a loss of spontaneity; the patient took no notice of his surroundings and slept during the greater portion of the day, or was semi-comatose in thirty-two of the cases.

There was loss of memory, mental failure, but the patient was cheerful in six cases.

The patient was suspicious and suffered from delusions, which were occasionally violent in one case.

The patient was irritable and violent in one case.

The patient was generally asleep and irritable when awake in two cases.

The patient was ambitious, excitable, and had loss of memory in one case.

There was slowness of mental processes, and the patient was simple and childish in one case.

There were mental anxiety, childishness, hallucinations, suicidal tendencies in one case.

The mental condition was not stated in five cases.

Seventeen of these lesions involved the right lobe, twenty-two the left, and eleven both lobes.

Ferrier² states the symptoms of disease of the frontal lobes as follows: Mental inactivity, forgetfulness, lack of judgment, decided change in character, irritability of temper and unusual stupidity, an inability to concentrate the attention, to think connectedly and continuously, to learn easily, to exercise self-control, and lastly, a state approaching mild dementia without delusions, in which the patient may become dirty and disregard all restraints of decency.

¹Brain, 1896, p. 346.

²Ferrier, quoted from Starr, Dercum's System of Nervous Diseases.

Starr³ says: "A decided mental change in character and disposition, a mental apathy and a mental somnolence must be regarded as a local sign of frontal lobe disease."

Mills⁴ says that in cases of disease of the prefrontal region, mental disturbances of a peculiar character occur, such as mental slowness and uncertainty, want of attention and control, and impairment of judgment and reason; closely studied the inhibitory influence of the brain both upon psychical and physical action is found to be diminished.

It must also be borne in mind that a brain tumor affecting any portion or lobe of the cerebrum causes mental changes, which are, however, not as pronounced as when the frontal lobe is affected.

In an analysis of fifty-six cases of brain tumor, I found mental indifference or apathy present in thirty-five cases, not present in one case, and not mentioned in twenty cases.

In an analysis of forty-two cases of brain abscess or cyst, I found mental symptoms present in twenty-seven cases, absent in four cases, and not mentioned in eleven.

Oppenheim⁵ considers mental apathy or somnolence of great importance in cases of brain tumor. The patient appears to be in a dreamy, sleepy state, and falls asleep while eating, or is even filthy in his habits, owing, of course, to the mental hebetude. The symptoms here narrated do not include those produced by pressure of the growth upon the neighboring areas as, for instance, the motor regions.

The mental condition of our patient was not such as to attract particular attention, until, perhaps, after the urination episode. It might then have been decided that the frontal area was involved, although the mental peculiarity at this time could have been attributable to the severe par-

³Starr, Dercum's System of Nervous Diseases.

⁴Mills, Dercum's System of Nervous Diseases.

⁵Lehrbuch der Nervenkrankheiten.

oxysm of pain. That none of the marked mental states found in prefrontal disease, such as those observed by different writers were present, was evident to us, and the symptoms which were observed were not different from those I have seen in cases where the tumor was located in the parietal or temporal lobe. It was, therefore, only by exclusion that a problematical diagnosis was possible, and then it was not of sufficient accuracy to warrant a surgical operation.

In Williamson's collection of fifty cases, four cases of abscess were all suitable for surgical interference. One case, MacEwen's, was operated on with success. Seven cases of tumor of the prefrontal lobes were so suitable and of such a moderate size that they could have been easily removed. In twenty other cases a tumor was removed; in one case successfully (Durante), in the others unsuccessfully.

Had an operation been undertaken the removal of so large a portion of the brain and the drainage of the cerebrospinal fluid would doubtless have produced a fatal termination soon after the completion of the operation. If it had been successfully removed the cyst in the left angular gyrus would still have been present and would have produced the same general symptoms, but perhaps not as severe. It would seem, therefore, that here, at least, was a frontal lobe tumor which would not have permitted a successful operative procedure.

DU TRAITEMENT DES NEURASTHENIES GRAVES PAR LA PSYCHOTHERAPIE (The Treatment of Grave Neurasthenia by Psychotherapy).
Dr. P. Valentine. (Bull. Gén. de Thér., Nov. 15, '96.)

It has already been proved by many authors that the mental treatment of neurasthenic subjects is most beneficial, especially in rebellious cases, which, as is well known, resist the usual methods. The author, considering the morbid psychical condition as the pathogenic element of the disorder, advocates treatment by hypnotic suggestion as the most reliable means, and enumerates three cases approaching the degenerative type of neurasthenia successfully treated. He concludes that psychotherapy in any form, with or without hypnosis, is the most successful method in obstinate cases, and that hypnotic suggestion never aggravates the cerebral disorder, but, on the contrary, invariably inspires the patients with stronger will power, more judgment and application.

MACALESTER.

Society Reports.

PHILADELPHIA NEUROLOGICAL SOCIETY.

November 22d, 1897. The President, Dr. Charles W. Burr, in the chair.

ON TABES DORSALIS "ARRESTED BY BLINDNESS."

This was the title of a paper read by Dr. Spiller. He stated that he used the word "arrested" because it had become well known in this connection through the writings of Dejerine, and had been applied to those cases in which the symptoms cease to progress, and even diminish in intensity after blindness appears. He quoted extensively from the statements of Dejerine on this subject, and reported two cases of this form of tabes, in one of which, however, the diagnosis was somewhat doubtful.

One patient had been gradually losing his eyesight during a period of eight years and had complained of pain in the lumbar region and shoulders. His gait was not truly ataxic, and Romberg's sign was not very evident. The patellar reflex in the right leg was much diminished in intensity, and in the left it was scarcely perceptible. Dr. Hansell reported that the patient had tabetic atrophy of both optic nerves. Argyll-Robertson's sign was not present, but the pupils were unequal. The case was interesting on account of the long duration of the gradually developing optic atrophy, without complete blindness even at the present time; the absence of distinct ataxia; the greater involvement of one side of the lumbar cord, as indicated by the difference in the knee-jerks; and the absence of Argyll-Robertson's sign.

The second patient, a negro, gave a clear history of syphilitic infection eight years ago. He had complained of pain in the lumbar region and one thigh, had been losing his vision for two years, and had a girdle sensation and disturbance of micturition. There was no incoördination of gait. The knee-jerks at the first examination were absent, but became visible after the administration of iodide of potassium. Dr. de Schweinitz found reflex iridoplegia, myosis, and divergence of the left eyeball. The optic nerves presented the signs of primary atrophy.

Dr. Spiller spoke of the difficulty of excluding cerebro-spinal syphilis in this second case, and of his inability to understand in what way Edinger's substitution theory could be applied to this form of tabes. He thought it would be well to avoid the name of locomotor ataxia as it is very awkward to speak of locomotor ataxia without locomotor ataxia.

Dr. William Osler:—I well remember that Dejerine called special attention to this very point, namely, the absence of ataxic symptoms in those cases affected with early optic atrophy, but I think that it is not quite correct to say that we do not see other instances of preataxic phenomena persisting for a long period, even years, without the development of ataxia; for instance, a localized paralysis of the ocular muscles. I have recently seen an exceptional case in which a diagnosis of locomotor ataxia was made in Paris in 1876. The patient has had lightning-like pains at intervals ever since. He has the Argyll-Robertson pupil and absence of patellar reflex, but he has no ataxia. In this case a diagnosis of locomotor ataxia in the preataxic stage has been made by most of the neurologists of Europe.

Dr. James Hendrie Lloyd:—Such cases are, of course, not unobserved. I have seen one or two, and I think that there is a case somewhat of this type at present in the Philadelphia Hospital. This man has complete optic atrophy, and so far has not the symptoms of posterior sclerosis. Four or five years ago he had several very curious congestive crises, much like what we see in dementia paralytica. Of recent years he has not had even these. He has a distinct history of syphilis.

Brissaud has divided locomotor ataxia into sensory and motor types, and has recorded one or two cases of the sensory type. I think he claims that the fulgurant pains are a common accompaniment of optic atrophy, and also that cases of the sensory type, with optic atrophy and fulgurant pain, without ataxia, are the cases most likely to present spinal arthropathies. This is a point which I am not prepared to confirm from my own observation.

With reference to theories, it may possibly be worth while to recall that the optic nerve-tract is not an ordinary peripheral nerve. It is analogous rather to a tract of the central nervous system. As Monroe has pointed out, it is allied in this way to the posterior columns of the spinal cord. It is probably for this reason that we find it degenerated along with the posterior columns in ordinary cases of tabes. It seems that in some persons the optic nerve-tract suffers first and more than the posterior columns of the cord from the same poison,—syphilis. The reason is obscure, but the fact remains. The only

trouble in reaching an explanation is that we do have some spinal symptoms, such as abolition of reflexes and fulgurant pains in these cases of the so-called sensory type.

Dr. Francis X. Dercum:—I think that the title "arrested by blindness" is objectionable. The facts would, perhaps, be better expressed by saying that after optic nerve atrophy appears the symptoms progress very slowly.

The second case which Dr. Spiller reported looks very much like a case of spinal syphilis; especially the return of the knee-jerk after the use of iodide is very suggestive. In the negro, further, locomotor ataxia is excessively rare. I have, however, had one case where the diagnosis was confirmed by autopsy. It is strange that the negro should enjoy such immunity from ataxia, while he does not enjoy a corresponding immunity from paresis.

Dr. Frederick A. Packard showed two brothers, aged ten and eleven years, respectively, out of a family of seven otherwise healthy children, with pseudo-hypertrophic muscular paralysis, changes in the thyroid gland and mental deficiency. The family history was unimportant, save for the existence of goitre, with nervousness and tachycardia—but without exophthalmos—in the mother, and the normal condition of the remaining members of their generation.

Dr. Francis X. Dercum:—Every now and then we see other types of nervous disease in children, which are either accentuated or first make their appearance after an attack of some infectious disease, such as measles. The three cases of cerebral spastic paralysis, shown by me before this Society last winter were an illustration of this fact. This appearance of accentuation after an infection is extremely interesting. It seems as though the power of resistance is occasionally so low in the nervous tissues—perhaps owing to some morphological peculiarity—that under so slight a cause as the toxicity of measles they undergo degeneration.

Dr. William Osler read a paper on paralysis of the hypoglossal nerve.

Dr. Charles K. Mills:—There is one point in Dr. Osler's paper which seems to me to be of special interest, and if I correctly understand the matter, it is doubtful whether his method of reference to accessorius paralysis is correct. In the light of recent observations, it seems to me questionable whether the nerve supply of the palatal and laryngeal muscles can

be regarded in any sense as from the spinal accessory nerve. The spinal accessory nucleus has been separated, and probably correctly, from the vagal nucleus and only supplies those muscles regarded as connected with the spinal portion of the nerve. The laryngeal paralysis would not then be referable to the accessorius.

Dr. Francis X. Dercum:—In this connection, I would allude to the morphological fact that in the orang-outang, the vagus supplies the larynx without fusing with the spinal accessory at all.

Dr. Charles W. Burr exhibited

A PORENCEPHALIC BRAIN.

The man from whom the specimen was removed was a negro, thirty-five years of age. Little is known of his previous history, except that he is said to have been an idiot and epileptic all his life. He came to the Philadelphia Hospital some years ago. He suffered from general convulsions and had a spastic gait and spastic rigidity of both arms, and such marked spasm of the tongue and lips that he could not talk. He remained in that condition some years. During the past summer he developed typhoid fever, from which he died.

At the necropsy two large symmetrical cavities were found in the brain, one in each hemisphere. Dr. Burr believed that these were of post-natal origin and regretted that the term porencephaly is used so indiscriminately for cavities in the cerebrum.

Dr. Purves exhibited for Dr. Mills, the brains from two cases of intraventricular hemorrhage. No convulsions had been observed in these cases at the time of the apoplectic seizure.

Dr. Charles K. Mills:—These are two interesting cases, although not of an unusual type. The first was of interest as regards diagnosis in connection with the history of the condition of the kidneys. Dr. Dercum and others in various papers, have called attention to the subject of hemiplegia in nephritis without gross brain lesion. This case presented many features that might have been explained in this way, but my experience has been that when such cases come to autopsy, it is rare not to find a lesion. The cases are also of interest from the absence of convulsions.

The post-mortem notes are worthy of special remark. It

is just as important in many of these cases to record the other vascular lesions that are present as it is to record the gross hemorrhage. In many cases of large, sudden, intracerebral hemorrhage lesions are found similar to those which are present in cases of concussion without gross hemorrhage. The symptoms which obscure the diagnosis in some cases are undoubtedly due to the numerous extravasations in various parts of the brain and membranes, and the secondary lesions which result in association with these.

A word with regard to autopsies in brain cases. My attention was called to the method of Dejerine by Dr. Sailer and Dr. Spiller, a year or two ago, and since then I have chiefly followed this method. In many necropsies, the methods adopted render the brain useless for subsequent microscopic and even macroscopic study. An old, and for many purposes, good method is that of Virchow. In this, after opening the ventricles, transections of the brain are made. This is a useful method for rapidly determining the conditions present, and if carefully pursued is a good one, especially for locating lesions in the fresh state.

Another method to which I first called attention is that of entering the brain from below, through the great transverse, calcarine and other fissures and keeping the callosum and parts above intact.

The method of Meynert is well known. In this city a method is sometimes used which seems to be a modification of that of Meynert. After the ganglia are exposed, an incision is carried around them deeply through the brain substance and necessarily through the temporal lobes. Horizontal or vertical sections are then made through the ganglia. This method is open to the objection of destroying the relation of the parts, making it impossible to subsequently examine the brain satisfactorily.

The method of Dejerine, which was described in the post-mortem notes of these two cases, is for many purposes a good one. A vertical incision is made, cutting off the occipital lobe. Another vertical incision may or may not be made in front of the basal ganglia. Then an incision is made about on a level with the callosum and the two hemispheres may be separated. In the method of Dejerine the cerebellum, pons and oblongata are removed as a separate specimen by an incision through the pons above the fifth nerve. One advantage of this is that the separate parts can be easily placed together.

A good method of examining the cerebellum is to cut off the lateral lobe on one side, and subsequently the lobe on the other side, keeping the middle lobe as a separate specimen.

Dr. Spiller:—These two cases of intraventricular hemorrhage are further proof of what I recently had occasion to

—speak of at the Pathological Society, viz., that convulsions are not always observed when hemorrhage into the ventricle occurs, and that they are of doubtful value in diagnosing such hemorrhage.

Dr. J. P. Arnold, by invitation, exhibited a

TUMOR OF THE CEREBRAL DURA.

The growth had its origin in the dura at the longitudinal fissure. It had displaced the cerebral tissue and interfered with the functions of the leg, arm and face centres.

Dr. James Hendrie Lloyd:—I saw this case in consultation with Dr. Stryker at the Presbyterian Hospital. A diagnosis of brain tumor had been made, but the question was as to the exact location. Looking at the specimen now, it is easy to regret that the man was not operated on. My own belief was that the tumor was in the antero-frontal region. The absence of convulsions, considering the location, is an interesting point. In the absence of distinct localizing symptoms I did not feel justified in recommending operation. There had been no distinct focal or signal epileptoid symptoms at any time. I inquired particularly into this point, and in the absence of such localizing symptoms I did not feel justified in concluding that the growth was in the motor zone. The mere hemiplegia was not enough to indicate this, because hemiplegia is seen in cases of brain tumor in the most widely separated regions. The patient had the peculiar mental inhibition that is sometimes seen in cases of brain tumor, especially in those of the frontal lobes.

Dr. F. X. Dercum:—I understand that the arm and face were more markedly affected than the leg. If so, it is probably to be explained by the way in which the tumor invaded the brain, *i.e.*, from the mesial surface outward, cutting across the fibres for the face and arm passing to the internal capsule. This case reminds me of one which I exhibited before this Society some years ago, in which the tumor had grown downward from the membranes, had separated the frontal from the motor convolutions, and had produced profound symptoms of a general character, but no convulsive seizures.

Dr. Spiller:—The condition of lock spasm which Dr. Arnold has described in this case, as an inability on the part of the patient to relax his hold on an object and a tightening of the grasp when he attempted to relax it, is like a symptom of Thomsen's disease. Schlesinger, of Vienna, told me that he had seen this sign in a case of syringomyelia, and Patrick has recently observed a similar form of muscular contraction in this same disease.

NEW YORK NEUROLOGICAL SOCIETY.

Stated Meeting, December 7, 1897.

B. Sachs, M. D., President.

TOXIC TREMOR AND HYSTERIA IN A MALE.

Dr. J. Arthur Booth presented a man whom he had seen one week ago at the French Hospital. The patient, sixty-one years of age, had been engaged up to 1885 as a mirror polisher, using mercury, and since then in the same occupation, but using silver instead. His family history was negative as regards nervous disease. At the age of twenty-two, he contracted syphilis, and was treated for this for some time. He then enjoyed good health up to thirteen years ago. At this time he fell on the street with both lower and upper extremities in a state of clonic spasm. He was taken to the Boston City Hospital. Some years later was under the care of Professor Charcot for several months. He returned to this country in 1887, and resumed his work of mirror polishing, using silver. He has had three or four attacks of tremor. The present illness began November 17, after having worked very hard for some time previously. The tremors presented by the patient, the speaker said, certainly resembled those observed in cases of mercurial poisoning. A very slight tap on the body would set up a pitiable degree of reflex tremor. There was no nystagmus, but there was slight ataxia. It was found that the tremor was greatly increased when attention was drawn to him, and for this reason it seemed not improbable that there was an hysterical element in the case. There were no bladder or rectal symptoms.

Dr. W. M. Leszynsky said that the case looked to him to be a functional one, added to the evident toxic tremor. The fact that he had such attacks, and had recovered from them, would seem to confirm this view.

Dr. Peterson and Dr. B. Sachs concurred in the opinion that there was marked hysteria imposed upon a mercurial tremor.

NEURITIS FOLLOWING AN INFECTED VACCINATION.

Dr. W. B. Noyes presented a baby. It had been vaccinated last spring, and the wound had become infected. When the bandages had been removed, it had been found that the child could not move the arm. Examination showed that the deltoid, biceps and the muscles supplied by the ulnar nerve were involved. They did not react to faradism, and gave the reaction of degeneration. The case seemed to him at the time to be a neuritis resulting from an infected wound, but two other opinions had been offered, viz., that it was the result of tight bandaging; and that the child had an attack of anterior poliomyelitis. The condition had developed within two weeks after the vaccination, and the motion of the arm had been largely recovered. There was no history of constitutional disturbance. His own opinion was, that the case was one of ascending neuritis involving the circumflex, ulnar and musculo-cutaneous nerves.

Dr. Peterson thought that poliomyelitis could hardly be considered. The diagnosis offered by Dr. Noyes might be correct, or else the condition was due to pressure. He inclined rather to the latter view.

PULMONARY TUBERCULOSIS AND TABES COMBINED.

Dr. Fraenkel presented a man thirty-nine years of age, unmarried, whose family history was negative. About ten years ago he contracted syphilis, but enjoyed fairly good health up to two years ago. Then he had repeated hemorrhages from the lungs, and developed rapidly the signs and symptoms of pulmonary tuberculosis. On admission to the Montefiore Home in May, 1896, there were pronounced physical signs of pulmonary tuberculosis, and of a large cavity at one apex, and death seemed near. The left pupil was but slightly responsive to light. The temperature never rose above 98.5 degrees Fahrenheit, and the pulse above eighty. He steadily improved up to May, 1897, at which time his sputum still contained tubercle bacilli, but his condition was in every way better. The signs of the cavity had disappeared. His pupils were unequal and the left was myopic. The scapular, abdominal, gluteal and plantar reflexes were exaggerated on both sides. The left patellar knee-jerk was markedly diminished. There

was no evidence of ataxia or inco-ordination. On November 18, 1897, physical examination showed the thoracic organs in the same condition. The patient then complained for the first time of occasional shooting pains in the left lower extremity. The sexual appetite was markedly diminished. The left knee-jerk was greatly reduced. The three points of interest were: (1) The marked unilateral character of the symptoms; (2) the combination of tuberculosis and tabes—a very rare condition; and (3) the mildness of both diseases.

Dr. Leszynsky said that he had seen a number of cases with various forms of disease, both of the lungs and abdomen, with but slight change of pulse and temperature, even in neurotic individuals. He had in mind a patient who showed very markedly unilateral pupillary symptoms such as had been referred to.

Dr. Noyes said that the case appeared to be one of fibroid phthisis. Non-progressive tabes is due rather to a collection of connective tissue than to any actual degeneration of the posterior columns. These two conditions were occasionally associated, and the case presented was probably an example of this.

TIC CONVULSIF.

Dr. Willam Hirsch presented a young man having Tic convulsif. He first saw him about a year ago, and the symptoms had not changed materially since then. The object in bringing the patient was to demonstrate a certain relation between his symptoms and the psychical condition present. On tapping the cheek, the patient made a number of grimaces. The fact that the symptoms had not undergone change was in marked contrast with what was observed in hysteria. This patient had had articular rheumatism, followed by endocarditis and the development of mitral insufficiency. Shortly afterward he had developed this neurosis.

WRY-NECK AND ASYMMETRY OF FACE.

Dr. Graeme M. Hammond presented for Dr. Meirowitz, a boy of sixteen, who had come under observation for the relief of what appeared to be a wry-neck. As far back as could be remembered the neck had been deflected

to the left, but he had never experienced any pain in the neck or elsewhere. There had been no twitching or jerking. Examination showed that the head deviated to the left; the left half of the face exhibited a perceptible slope from above downward; the left eyebrow, ala nasi, angle of mouth and angle of jaw were all higher than the corresponding points on the right side. The muscles of the neck were distinctly hypertrophied. The skull over the parietal region showed a perceptible flattening, not observed on the other side. The movements of the head were free in all directions. The hair curved to the left side. There was no disturbance of the cranial nerves, and no hemiatrophy, and the intelligence was normal. There was no family history of similar defects.

Dr. Fraenkel remarked that cases of congenital wry-neck had been explained as resulting from a lessened developmental resistance on one side.

Dr. Peterson remarked that it was unusual to find so good an example of the unequal development of the ear.

Dr. Onuf said that in most cases of congenital wry-neck there was this asymetry of the face. He was not sure that the pressure was alone responsible for the unequal development; it would hardly explain the asymmetrical development of the ear. The atrophy seemed to him possibly the result of twisting of the carotid artery on one side, with a consequent interference with the nutrition on that side.

ENORMOUS HYDROCEPHALUS.

Dr. F. Peterson presented the brain from such a case, one of the largest for many years at the Randall's Island institution. The patient was a woman of twenty who, in the early stages, had had a left hemiplegia and imbecility. As the disease progressed, she became diplegic and completely idiotic. The following are the measurements of the head: Circumference 63.5 cm.; approximate volume 1,714 cc.; naso-occipital arc 47 cm.; naso-bregmatic arc 16.5 cm.; bregmatic-lamboid arc 19 cm.; binauricular arc 50 cm.; antero-posterior diameter 19.5; greatest transverse diameter 18.5 cm.; length-breadth index 94.8 per cent.; binauricular diameter 12 cm.; auriculo-bregmatic radii 20 cm.; facial length 11.2 cm.; empirical greatest height 17.7 cm.; height Beta-x 19.2 cm. At the autopsy the sutures were found fully united and the fontanelles perfectly closed. The skull bones averaged 8 mm. in

thickness. The dura was not adherent at any part. There was a very little fluid in the dura. All of the fluid was in the ventricles of the brain, more on the right side than the left, for the cortex of the right hemisphere had become in part membranous. Five pints of clear serum were removed from the ventricles. The third and fourth ventricles partook, to some extent, in the dilatation. It was unfortunate that through a mishap the brain was so injured that the patency of the foramen of Magendie could not be determined. Dr. Peterson said that, in his experience, it was difficult to examine this foramen, and apparently impossible to demonstrate the presence in any human being of the foramina of Mierzejewsky. It was very important in all cases of chronic hydrocephalus to examine, if possible, the foramen of Mierzejewsky. It was much easier to determine the patency of the aqueduct of Sylvius and of the foramen of Monroe. He had, with Dr. Blake, at the Anatomical Laboratory of the College of Physicians and Surgeons, examined the brain of a ten-year-old hydrocephalic of Randall's Island, and in this instance, at least, had determined definitely the permeability of the foramina mentioned, and the aqueduct.

ABSCESS OF THE BRAIN.

Dr. Peterson presented a specimen showing an abscess of the brain that had been operated upon. The case had been seen on September 3, 1897, in the service of Dr. Brown, of the Mountainside Hospital, at Montclair, N. J. The patient was a male, forty-one years of age, who, in an attempt at suicide, had struck his head upon a rusty spike and had sustained a compound depressed fracture in the right occipital region, very near the middle line. He was trephined, and the depressed bone removed. The dura was found uninjured, and was left untouched. The wound became slightly infected, but aside from slight fever no symptoms of importance appeared until two days before he had seen the man, that is, for about two weeks after operation. He then gradually developed in twenty-four hours a left hemiplegia, with left hemianaesthesia. Examination showed complete paralysis of the left arm and leg, with considerable anaesthesia over the paralyzed limbs; slight analgesia in places, and hyperalgesia in oth-

ers. The plantar and cremasteric reflexes were normal. The knee-jerks were subtypical, slightly greater on the left side. The mind was clear. There was no aphasia, no involvement of the face or tongue. There was no evidence, in pupils or pulse, of intra-cranial pressure, and no symptom of cortical irritation. The temporal half of the field of vision of the left eye was lost. The right seemed normal. He heard a watch at only half the distance with the left ear as compared with the right. Taste was normal on both sides of the tongue. The fundus seemed slightly cloudy. A diagnosis was made of abscess deep in the brain, in the region of the posterior limb of the internal capsule. The surgeon was advised to trephine in the parietal region, considerably back of the motor area, and insert an exploring needle down into the supposed site of the abscess, should the patient grow worse. Two days later this was done. The abscess was found at the site suggested, and was drained. The temperature became normal, the anaesthesia disappeared and the patient moved his left hand, but a few days later he suddenly became worse, and died. The brain was sent to Dr. Peterson for examination. He found the dura very sclerotic over the site of the original injury close to the superior longitudinal sinus. The convolutions of the right hemisphere were considerably flattened. There was no meningitis. Deep in the interior of the right hemisphere was an abscess, about the size of a hen's egg.

HEMIATROPHY OF THE BRAIN.

Dr. Peterson then presented a specimen showing apparent hemiatrophy of the brain. The brain was that of a Randall's Island patient, a man of thirty years, who had been in the idiot asylum for several years. A history of the origin of his trouble could not be obtained. He was a large, heavy man, with a hemiplegia (the arm being much worse than the leg), and with frequent and severe general epileptic convulsions. While able to be about, he was dull and stupid, and in intelligence would be placed in the grade of moderate idiocy. He died in an epileptic fit. There was nothing abnormal in the other organs of his body, but the brain presented the condition of marked right hemiatrophy. There was very slight evidence of

microgyria, the convolutions differing as regards normality from those of the opposite side in being somewhat smaller. The membranes over the right hemisphere were, perhaps, somewhat thicker than over the left. An examination of the vessels at the base of the brain showed that the blood supply was apparently equal for the two sides. It was undoubtedly a lack of development through some obscure and early pathological process.

A CASE OF AMYOTROPHIC LATERAL SCLEROSIS—SUPPLEMENTARY REPORT.

Dr. William Hirsch said that on October 1, 1895, he had presented to the Society a case of this kind. The patient was forty-three years of age and had had poliomyelitis at the age of twenty. The clinical symptoms were such, that at that time he had traced them from the old scar in the left anterior horn in the cervical region. The process must have approached the left pyramidal tract, and then after affecting the right horn, spread over to the right side. That was his opinion at that time, and a few such cases had already been presented. In the discussion, it was claimed that the case was one of ordinary progressive muscular atrophy. But further clinical observation of the case showed that this was not the case. The man died on June 3, 1897, and Dr. Hirsch was now able to present microscopical specimens of the spinal cord in different regions, proving the correctness of his diagnosis. He said that in these specimens one could see the connective tissue spreading from the anterior horn to the left pyramidal tract, and the latter converted into connective tissue. The cells in the anterior horns had been destroyed, the connective tissue had formed all through the lateral tract. In the cervical region one bundle of connective tissue had spread backward into the posterior columns, as he had previously diagnosticated. Deep degeneration of the pyramidal tracts could be followed down to the lumbar region.

Dr. Hammond asked how he would differentiate these specimens from progressive muscular atrophy.

Dr. Hirsch replied that the specimens showed that the case was not one of ordinary degeneration of the lateral tracts, but that the connective tissue grew horizontally from the anterior

horn to the side. Furthermore, the ordinary progressive muscular atrophy is a symmetrical disease.

Dr. G. M. Hammond said that in three unquestioned cases of progressive muscular atrophy that had been under his observation, there had been a typical degeneration of the pyramidal tracts. On looking up the subject, he had found that Gowers made the statement that he had not seen a case of progressive muscular atrophy in which the pyramidal tracts had not been extensively affected. It was true that in the specimen presented, one horn was affected more than the other, nevertheless both horns were involved.

Dr. C. L. Dana thought these sections looked very much like specimens from a case of progressive muscular atrophy. If the pathological change were not one of degeneration of the horns and lateral columns, he would like to know what Dr. Hirsch thought it really was?

Dr. B. Sachs thought the main point was as to whether the degeneration was strictly bilateral in the lateral columns, or more marked on the side of the old poliomyelitis.

Dr. Dana thought that at the time of the poliomyelitis there had been considerable hemorrhage in the neighborhood of the anterior horns, and the lateral column might have been injured and a scar formed. The degeneration would then be secondary to this scar.

Dr. C. A. Herter said that the fact that the degeneration of the lateral tract was chiefly on one side pointed very strongly to the local nature of the trouble, and opposed the idea of the case being one of progressive muscular atrophy.

Dr. Hirsch said that it was not a descending degeneration, but a growing out of connective tissue from the anterior horns peripherally and laterally in the direction of the pyramidal tracts; and the series of sections that he had made enabled one to follow this branching out of the bundles of connective tissue. This was quite different from an ordinary case of progressive muscular atrophy complicated by a secondary degeneration of the lateral tracts. The specimen presented showed that an old focus in the anterior horn had set up a secondary inflammation. It was difficult to form a correct conception of the case without a careful examination of the whole series of specimens. The case represented one of those processes of inflammation characterized by the formation of connective tissue.

Dr. M. Allen Starr asked Dr. Hirsch if the focus of inflammation in one anterior horn and extending into the adjacent columns might not produce a secondary sclerosis downward through the cord, and so explain the condition.

Dr. Hirsch replied that it extended laterally as well as upward. He had not yet examined the medulla oblongata. The

poliomyelitis was in the cervical region of the cord, about the region of the left arm.

UNILATERAL ATROPHY OF THE BRAIN.

Dr. Pearce Bailey reported a case of this kind occurring in a carpenter who was fifty-seven years of age at the time of his death. He had been perfectly healthy up to the forty-seventh year, and had then had an attack of apoplexy. At this time he awoke to find one side powerless, and this power he never regained. The picture was that of extreme left hemiplegia. The atrophy in the paralyzed limbs was not unusual in degree. There was no disturbance of psychical sense functions or general intellectual capacity. Speech was clear. In the summer of 1897, he died of acute lobar pneumonia. At the autopsy, the dura mater was found adherent at the right anterior portion of the cranial border, and in the adhesions between the dura and pia was a collection of clear serum. The left hemisphere was normal in size. The whole right hemisphere was much smaller than the left. The unilateral atrophy involved also the brain stem and spinal cord. The right internal carotid artery was less than half the size of the left. The forward portion of the right hemisphere was in a state of advanced chronic softening. Sections from the pre-central gyrus and from the occipital lobe contained few ganglionic elements. The cuneus was affected similarly, though to a lesser degree. Although the frontal lobes, which are supposed to be intimately connected with mental processes, were almost completely obliterated on one side, mentality had not been impaired. It was believed that the lesion occurred after complete cerebral and bodily growth had been attained. It was assumed that the original lesion was a thrombus in the right internal carotid artery, at or near its bifurcation. This case emphasized the necessity for caution in regarding any single region of the brain as indispensable to mental action.

Dr. Herter said that he had no doubt that the extreme degree of atrophy was the result of extensive arterial lesion; possibly the very slow development of the vascular lesion in the frontal region would go far towards explaining the absence of marked cerebral symptoms. In his experience, comprising cases of acute softening and abscess, where there

had been extensive destruction of the frontal lobe on one side, there had been considerable interference with the mental functions.

Dr. Dana said that it was remarkable how the faculty of language could be maintained notwithstanding extensive destruction of the right side of the brain.

Dr. Sachs said it seemed to him very evident that Dr. Bailey's case was distinctly vascular in origin, which was in sharp contrast with Doctor Peterson's case.

Dr. Bailey, in closing, said that there seemed to be no doubt that the entire half of the brain in his case had been affected.

A NEW DEVICE FOR ADAPTING THE INCANDESCENT ELECTRIC CURRENT TO THERAPEUTIC PURPOSES.

Dr. A. D. Rockwell demonstrated the action of an apparatus which, he said, comes under the head of shunt controllers, but is of novel construction. It ranges from 1 to 75 volts, and can be varied one volt at a time up to 30 volts, and then 3 volts at a time for the balance. The galvanic current in this way can be readily superimposed upon the faradic, giving most interesting therapeutic results. The movement of the milliamperemeter during the simultaneous passage of the two currents is somewhat greater than when only the galvanic current passes, probably owing to a lowering of the resistance of the body by the faradic current. This combined application had been found by him particularly useful in cases of exophthalmic goitre.

NEUROLOGICAL FRAGMENTS. Hughlings Jackson (Lancet, Jan. 2d, 1897).

In this continuation of his series of papers, termed "Neurological Fragments," Hughlings Jackson gives us a consideration of cervical fracture-dislocation. It is an extremely interesting article dealing with symptoms "at the seat of lesion" and "below the seat of lesion," and the effects upon the four systems, namely, the thermal, the respiratory, the circulatory and the digestive. Incidentally certain symptoms from the side of the sexual system are also considered. Jackson indulges in speculation concerning the severance of intrinsic and extrinsic fibres, in observations on extremely high temperatures resulting from lesions in this regions (cervical) and upon the effects of the cutting off from the highest, middle and lowest levels (centres).

It is almost impossible to adequately review the paper in question without entering into details, which would involve long quotations from the original. It would be better to read the article in *to*to.

STERNE.

Periscope.

With the Assistance of the Following Collaborators:

CHAS. LEWIS ALLEN, M.D., Wash., D.C. R. K. MACALESTER, M.D., N.Y.
J. S. CHRISTISON, M.D., Chicago, Ill. J. K. MITCHELL, M.D., Phila., Pa.
A. FREEMAN, M.D., New York. H. PATRICK, M.D., Chicago, Ill.
S. E. JELLIFFE, M.D., New York. JOSEPH SAILER, M.D., Phila., Pa.
WM. C. KRAUSS, M.D., Buffalo, N.Y. HENRY L. SHIVELY, M.D., N.Y.
W. M. LESZYNSKY, M.D., New York. A. STERNE, M.D., Indianapolis.

ANATOMY.

- 28 NOUVELLES RÉCHERCHES SUR LA STRUCTURE FINE DE LA CELULE NERVEUSE (New Researches upon the Minute Anatomy of the Nerve Cell.) S. Marinesco (La Presse Médicale, 1897, No. 49).

In this article, a continuation of one previously reviewed, the author describes the lesions produced in the cells of the spinal ganglia and anterior horns, by alcohol, arsenic, and the hydrophobic virus. These lesions consist of a chromatolysis, most generally peripheral, but sometimes diffuse, differing somewhat with the different poisons.

Basing his conclusions upon the results of examination, and comparison of normal with pathological cells, he discusses the fine anatomy and probable physiology of the nerve cell. The nerve cell is composed of three essential elements—1. the chromophile substance; 2. an incolorable "figured" substance; 3. an incolorable amorphous substance, which binds the chromophile granules together. To the second of these, attention is specially called. It consists of a number of very fine fibrils, which cross and recross, making up a network, in whose interstices the chromophile granules are placed. The meshes of this network vary in fineness. To it the name of "spongio-plasma" is applied. Its structure is best shown in cells which have undergone chromatolysis. As to the relation of the fibrils of the axis cylinder process and the protoplasmic processes with the fibrils of the spongio-plasma, the author thinks that they are directly continuous. With regard to the chromophile substance he takes issue with the authors who regard it as having only a nutritive function. For him it is an unstable chemical compound, and serves as a reservoir for the storage of nerve energy. "Kineto-plasm" he calls it. Being unstable, it breaks up easily to enter into new combinations, and in so doing liberates energy, which reinforces the current which has caused the explosion. It may also be caused to liberate energy by the direct action upon it of poisons, as strychnine, etc. Other poisons destroy it at once, producing paresis or paralysis.

ALLEN.

29. ZUR KENNTNISS DER CEREBROSPINAL FLÜSSIGKEIT (The Cerebrospinal Fluid.) H. Nowvatzki. (Deutsche med. Wochenschrift, 1897, No. 2.)

Nowvatzki has made a study of the cerebrospinal fluid, and states that in healthy calves the chemical constituents are as follows: .046-

per cent. grape sugar, .01—.03 per cent. albumen, .28 per cent. organic constituents, 1.07—1.1 per cent. of dry residuum, .7—.8 per cent. ash. There were no albumoses or peptones in the organic constituents, and the albumenoid was a globulin.

In the cerebrospinal fluid of paralytics, .5 per cent. grape sugar was found, which amount, however, diminished rapidly after death.

JELLIFFE.

30. ÜBER DIE ANWENDUNG ELEKTIVER FÄRBENMETHODEN AM IN FORMOL GEHÄRTETEN CENTRALNERVENSYSTEM (Formaline as a Fixative for Nervous Tissues.) Gudden (Neurologisches Centralblatt, 16, 1897, p. 24).

Gudden gives the results of experiments on staining nervous tissues which have been hardened in formaline. With such he uses the Nissl staining methods. The material to be used for the Weigert methods requires a preliminary placing in $\frac{1}{2}$ per cent. chromic acid for ten hours, at the temperature of the room to prepare it.

JELLIFFE.

31. A MODIFIED FIXING FLUID FOR GENERAL HISTOLOGICAL AND NEURO-HISTOLOGICAL PURPOSES. A. P. Ohlmacher (Journal of Experimental Medicine, 2, 1897).

After experimenting with various modifications of Carnoy's fluid, which is so highly recommended by Van Gehuchten, the author presents the following modifications:

Anhydrous alcohol.....	80 pts.
Chloroform	15 "
Glacial acetic acid.....	5 "
Corrosive sublimate.....	to saturation.

The alcohol employed is made by dehydrating 95 per cent. alcohol with anhydrous copper sulphate. About twenty grammes of powdered corrosive sublimate are required to slightly over-saturate 100 ccm. of the fluid.

Ordinary pieces of tissue are sufficiently fixed in fifteen minutes to half an hour. For the brain, after subdivision, eighteen to twenty-four hours are usually sufficient. Penetration is rapid, and the cytologic details are said to be excellent.

JELLIFFE.

32. EINE NEUE METHODE ZUR FÄRBUNG DES CENTRALNERVENSYSTEMS (A New Method of Staining the Nervous System.) J. Allerhand (Neurolog. Centralblatt, 16, 1897, p. 727).

The author describes a method whereby materials which have been hardened in a great variety of fixative fluids, including alcohol, can be used in the Weigert-Pal technic. He uses the Liquor ferri sesquichloratis of the German Pharmacopoeia and a preparation of tannic acid:

Thin sections are stained for from fifteen to twenty minutes in a fifty per cent. solution of the iron; after some washing in water the specimens are transferred to a twenty per cent. solution of tannin, which is specially prepared; the tannin being dissolved in distilled water and boiled, and then left in an open flask in the sunlight where, after a length of time, moulds develop; after two or three weeks the fluid is filtered and is then ready for use. The specimens, after treatment in the iron, are placed in the tannin solution, and for from one to two hours, stay at a temperature of about 50 deg. C. They can be differentiated by the regulation Pal methods.

JELLIFFE.

PATHOLOGY.

33. ÜBER DEGENERATIONSSHERDE IN DER WEISSEN SUBSTANZ DES RÜCKENMARKS BY LEUKÄMIE (The Spinal Cord in Leukemia). M. Nonne (Deutsche Zeitschr. für Nervenheilk., 10, 1897, p. 167).

To Nonne we are already indebted for much valuable information regarding the changes in the spinal cord induced by pernicious anemia and the symptoms resulting therefrom. He now contributes two cases of cord disease caused by leukemia, a knowledge of which seems heretofore to have been confined to a single case reported by W. Müller (Inaugural Thesis, Berlin, 1895).

The first case was a man aged 59, whose symptoms began six months before death with general debility, dizziness and anorexia. On examination two months after the inception of the disease, the liver and spleen were found to be greatly enlarged, the red blood corpuscles numbered 1,896,000, the white 630,000 to the cubic millimeter, and the percentage in hemoglobin was only 40. The bones were nowhere tender to pressure, and glandular enlargement was limited to a few glands in the groin and axillæ, some of which were as large as a filbert. Sensation, motion and the reflexes were normal. While under observation the number of red blood corpuscles increased slightly, and the spleen diminished in size, but the patient suffered from large intermuscular hemorrhages and died of marasmus and catarrhal pneumonia. Microscopic examination of the cord revealed small myelitic foci or, more strictly speaking, foci of acute or subacute nerve degeneration, scattered through the white substance from the upper lumbar region to the medulla oblongata. Some of these degenerated points were large enough to be seen by the naked eye, and all the stages of degeneration were to be observed from a simple puffed-up appearance of the myeline sheath and swelling of the axis cylinder, to segmentation, breaking up and disappearance of the nerve fibres with compensatory hypertrophy of the neuroglia. Changes in the vessels, hemorrhages, cellular infiltration and extravasation of leucocytes were entirely wanting, and the gray matter throughout with the nerve roots was absolutely normal.

The second patient, a man aged 31, noticed first some enlargement of the abdomen, and during the next two months marked general weakness developed. Irregular febrile movement then appeared, and the patient was admitted to the hospital where he was found to have an enormous spleen, reaching to the right inguinal region. The lymphatic glands were not enlarged, and the bones were not noticeably tender, but examination of the blood showed a great increase of white corpuscles, viz., 1,940,000 red, and 910,000 white, to the cubic millimeter. Objective symptoms of involvement of the nervous system were wanting. The patient died eight days after admission to the hospital and four after the appearance of the first symptoms noticed. A careful microscopic examination of the spinal cord revealed lesions identical in character, size and distribution with those of the first case, the gray matter, nerve roots and vessels being intact. It should be added, however, that the Nissl method was not employed as the material was hardened in Müller's fluid, and hence minute changes in the nerve cells, were not to be positively excluded. PATRICK.

34. EIN BEITRAG ZUR KENNTNISS DER IM VERLAUFE DER PERNICIÖSEN ANÄMIE BEOBSACHTETEN SPINALERKRANKUNGEN (Changes in the Gray Matter of the Spinal Cord in Pernicious Anemia). Teichmüller (Deutsche Zeitschr. für Nervenheilk., 8, 1896 p. 385). The author reports a case of pernicious anemia with arterio-

sclerosis, chronic enteritis, paresthesia, and increased knee-jerk. On post-mortem, small hemorrhages were found in the corpora quadrigemina and corpora striata. Microscopic examination showed changes such as have been described in the posterior columns, and, in addition, hemorrhages in both the white and gray matter, with degeneration in the anterior and lateral columns. The author combats the idea that the changes in the cord in pernicious anemia represent a combined system of disease, and looks on the changes of the gray matter as of chief importance.

The paper is illustrated with photo-micrographs, and a bibliography is included. VOGEL.

35. THE SPINAL CORD IN PERNICIOUS ANEMIA. Clarke (British Medical Journal, 1897, p. 325).

The author reports two cases. In the first, the patient being a woman of 46 years, the changes in the cord were substantially those hitherto described as occurring in pernicious anemia; i. e., degeneration of the posterior columns with some involvement of the lateral columns. The posterior column degeneration was very intense, included most of the columns of Goll and Burdach and extended from the highest to the lowest point of the cord. Lateral column changes were limited to the pyramidal tract of one side in the lower dorsal and upper lumbar regions; the direct cerebellar tract remained intact.

Changes in the second case, a man of 38, were decidedly exceptional, the degeneration of white matter being restricted to small symmetrical patches just external to the gray matter between the anterior and posterior horns. These areas were also limited in longitudinal extent, corresponding to about one cord segment. In the gray matter were marked changes which seemed to be contrary to the rule.

"The vessels were intensely injected, and there were many hemorrhages, varying in size, but all microscopic, into the grey matter. These hemorrhages were distributed chiefly about the central parts of the gray matter, posterior part of the anterior cornua and neighborhood of the commissure. Besides the hemorrhages there were in places small areas in which the gray matter was granular, partly disintegrated or sclerosed. The nuclei of the glia cells were either normal or slightly increased in number.

Certain of the nerve cells of the anterior cornua were swollen, opaque and homogenous, their nuclei obscured; others were highly granular and deeply pigmented, and a few appeared small and shrunken, but the large majority appeared normal. The walls of the small vessels were much thickened, and very many showed hyaline change. In places the anterior fissure was broadened by the distended arterial branches. The central canal was blocked. These changes in the gray matter were judged to be of fairly recent occurrence, and not of old standing, and were most marked in the upper dorsal region. No hemorrhages were noticed in the other organs post mortem."

PATRICK.

36. ANATOMISCHE UND EXPERIMENTELLE UNTERSUCHUNGEN ÜBER DIE RÜCKENMARKSVERÄNDERUNGEN BY ANÄMIE. (Anatomical and Experimental Studies in the Changes of the Spinal Cord in Anemia). G. V. Voss (Deutsches Archiv für klin. Med., 58, 1896, p. 489).

By the injection of such agents as pyrocin, glycerin, pyrogallol, and tolylendiamin, the author induced an artificial anemia, which, although not of the genuine pernicious form, still presented the fol-

lowing symptoms; diminished number of red blood cells, decreased amount of hemoglobin, poikilocytosis, fatty hemorrhage. Although the animals were maintained in this condition for some time, no typical lesion of the spinal cord could be discovered. The negative results reached inclined the author to the belief that the disease in man is due to as yet unknown chemical poisons. VOGEL.

37. LE SCLEROSI COMBinate DEL MIDOLLO SPINALE NELLE ANEMIE PERNICIOSE. (Combined Sleroses of the Spinal Cord in Pernicious Anemia). G. Bastianelli (Bulletino della R. Accademica Medica di Roma, 22, 1896).

The author groups the observations that have been made on combined scleroses of the cord in pernicious anemia under two headings. The first class embraces those cases in which the anemia is the most prominent factor, and the nervous symptoms are slight, the disease running its course in a comparatively short time. The cord presents irregular lesions, situated principally in the posterior columns. Into the second class are put those forms in which the nervous manifestations are the predominating feature of the clinical picture, and the pernicious anemia is a supervening complication. Here the spinal lesions are the same as those found in other primary combined scleroses, and are quite in accord with the nervous symptoms. The author does not believe in a direct relation between the anemia and the spinal lesions, for the changes of the chord induced by anemia alone are simple swelling of neuroglia and nerve fibres and not a form of degeneration. The theory of Minnich that vascular lesions are capable of producing degeneration of the white matter of the cord, is not satisfactory, for similar conditions are present in other affections of the cord, and the author also found perfectly normal vessels in instances of this disease. The most likely explanation is the presence of some toxic agent capable of producing both diseases, and which the author believes frequently to be of interstitial origin. VOGEL.

38. DES ACCIDENTS NERVEUX OBSERVÉS DANS L'ANEMIE PERNICIEUSE ET LEUR PATHOGENIE. (Nervous Lesions in Pernicious Anemia and their Pathology). Annales et Bulletin de la Société de Médecine de Gand, 1897, pp. 139-147.

This article contains a short resumé of the more recent work that has been done in pathological lines upon nervous tissues in this disease.

39. CONTRIBUTION A L'ETUDE DES LESIONS MEDULLAIRES DANS L'ANEMIE PERNICIEUSE PROGRESSIVE PROTOPATHIQUE ET DANS LES L'ANEMIES SYMPTOMATIQUES DE L'ADULTE (Medullary Lesions in Pernicious Anæmia). E. LeNoble (Revue de Médecine, 1897, p. 425).

The author considers two cases of pernicious anæmia, the first occurring in a young man of twenty-six years of age, whose blood showed typical changes. The nervous symptoms were vertigo, tinnitus, ocular disturbances, frequent headaches, slight tremor of the hands, a marked hyperesthesia of the skin and greatly increased knee-jerks.

The pathological changes observed in the nervous tissues were old hemorrhages in the bulb, and disseminated and extensive hemorrhages in the cord. The second case was one of symptomatic secondary anæmia without any marked clinical disturbances. The pathological findings were negative. JELLIFFE.

CLINICAL NEUROLOGY.

40. L'EXAGÉRATION DES RÉFLEXES ET LA CONTRACTURE CHEZ L'HEMIPLÉGIQUE. (The Exaggeration of the Reflexes and the Contracture in the Spastic and the Hemiplegic). A. Van Gehuchten (Journal de Neurologie et d'Hypnologie, 2, 1897, p. 621).

A lesion of the pyramidal tract in its cerebral portion causes flaccid paralysis and loss of voluntary movements, whereas a lesion in its spinal portion causes spasticity without paralysis, but with impairment of voluntary movements. According to Van Gehuchten, the motor cortex is connected by two tracts with the cells of the peripheral motor fibres; by a direct corticospinal tract, and by a corticopontocerebellospinal tract. These two tracts are united from the cerebellar cortex to the lower part of the pons, and in the latter region they separate, one passing to the cord as the pyramidal tract, and the other to the cerebellar cortex, from which point other fibres descended into the anterior part of the lateral column of the cord. The flaccid paralysis and the loss of voluntary motion in the hemiplegic are the results of destruction of both tracts. The spasticity and partial preservation of voluntary motion in the spastic are the result of interruption of the corticospinal tract, with preservation of the corticopontocerebellospinal tract. The interruption of the corticospinal tract within the cord suspends the inhibitory action of the cortical cerebral cells upon the motor spinal cells, and is the immediate cause of the exaggeration of the reflexes, whereas the motor tract, passing through the cerebellum, preserves the connection with the cerebral cortex and prevents paralysis. It has been thought that interruption of the pyramidal tract causes paralysis, and that the degeneration of these fibres causes spasticity or contracture. Van Gehuchten disputes these statements, and attempts to show that the theories advanced in regard to the effects of secondary degenerations cannot satisfactorily explain the contractures.

Babinsky has recently stated that there is impairment of the muscular tonus in hemiplegia, *i. e.*, that the passive movements of extension and flexion are greater in the paralyzed limbs. This hypotonicity has been noticed not only in recent cases, in which there had been no exaggeration of the tendon reflexes, but also in cases of hemiplegia which had lasted several months, and in which there had been exaggeration of the tendon reflexes.

The muscular tonus is the outward expression of the condition of the stimulation of the spinal cells, and this condition is the result of stimulation and inhibition by means of the posterior spinal roots, and the pyramidal and descending cerebellar fibres. The muscular tonus is exaggerated in the spastic because of the stimulation through the cerebellospinal and posterior root fibres, and the loss of inhibition through the pyramidal fibres. Complete transverse lesions of the cord destroy the cerebellospinal and the corticospinal fibres and cause hypotonia. The hypertonia produced by destruction of these two tracts in cerebellar hemiplegia proves that the hypotonia in complex lesions of the cord is not due merely to destruction of fibres from the cerebellum, together with the pyramidal fibres, but to destruction of the corticopontocerebellospinal tract, together with the pyramidal. The exaggeration of the reflexes in both the hemiplegic and the spastic, and the impairments of the muscular tonus in the former and the exaggeration in the latter, prove that the condition of the reflexes is independent of the muscular tonus. This Van Gehuchten believes, because:

1. In the hemiplegic there is impairment of the muscular tonus and exaggeration of the reflexes.

2. In the spastic there is exaggeration of the muscular tonus and exaggeration of the reflexes.

3. In the cerebellar lesions there is exaggeration of the reflexes and normal muscular tonus.

4. In complete transverse lesion of the cervicothoracic cord there is diminution of the muscular tonus and abolition of the reflexes.

The cerebellum seems to have an important influence on the condition of the reflexes, if not on the tonus, for lesions which destroy both motor tracts in the cerebrum cause an exaggeration in the reflexes, whereas lesions which destroy both tracts in the cord cause an abolition of the reflexes.

The contracture of the hemiplegic is not comparable with the contracture of the spastic. In the former there is a lesion of both motor tracts, in the latter only of the corticospinal fibres. The contracture of the hemiplegic is late in its manifestation; it is always preceded and accompanied by paralysis; it presents a variable location in the muscles of a limb; the muscular tonus is impaired, and the contracture is persistent in repose as well as in activity. The extensors of the upper limb are usually more paralyzed than the flexors, and all voluntary efforts to move the paralyzed member affect the flexors. The posthemiplegic contracture is the result of muscular contraction, not of retraction. The variations of type of the contracture depend on the fibres destroyed in the internal capsule.

The contracture of the spastic begins as soon as there is destruction of the spinal portion of the pyramidal tract; it is not associated with complete paralysis. It involves all the muscles of the affected limbs; the muscular tonus is exaggerated and the degree of the contracture is increased by motion. The contracture of the spastic is the clinical expression of the exaggeration of the muscular tonus.

Van Gehuchten makes the following brief statements:

1. The secondary degeneration of the pyramidal fibres and the consecutive sclerosis are not manifested by any outward signs.

2. The exaggeration of the reflexes and the contracture in the hemiplegic and in the spastic are independent of the pathological conditions.

3. The condition of the reflexes is entirely independent of the condition of the muscular tonus.

4. The normal condition of muscular tonus depends, at least in great part, on the connection of the spinal with the cortical cerebral cells. Complete separation of these causes diminished tonicity.

5. The normal condition of the reflexes also depends on this connection of the spinal with the cortical cerebral cells, and complete separation of these causes exaggeration of reflexes. The condition of the reflexes in complete, transverse spinal lesions (Bastian) cannot be satisfactorily explained.

6. The contracture of the hemiplegic is very different from the contracture of the spastic.

7. The contracture of the spastic is of central origin and is due to exaggeration of the muscular tonus. This exaggeration is due to interruption of the corticospinal tract with preservation of the corticopontocerebellospinal tract.

8. The posthemiplegic contracture is of peripheral origin, and is due to a minor degree of paralysis in the flexor muscles of the upper limb and to the contraction of these.

There are fibres in the anterolateral columns which degenerate downward after lesions of the cerebellum.

SPILLER.

PSYCHOLOGY AND PSYCHIATRY.

41. COMPARATIVE OBSERVATIONS ON THE INVOLUNTARY MOVEMENTS OF ADULTS AND CHILDREN. M. A. Tucker (*American Journal of Psychology*, 8, 1897, p. 394).

The author, during his experiments, made the thought of the reagent a "constant" by some simple exercise of the mind. A slight modification of Jastow's automatograph was used, the finger tip resting slightly upon it. A circle being regarded as the field of motion for the hands, it was divided into an upper half, or positive field, and a lower half, or negative field; and the right and left halves were called the right and left fields.

In the first series of experiments the reagent's (adults 18, children 13) attention was directed to some stationary object. It was found that for adults 55.5 per cent. of the movements were into the positive or forward field, while 48.5 per cent. were in the negative field. For children the entire movements in the positive field were only 26.9 per cent., while those in the negative field were 73.1 per cent.

In those whose constant attention (18 adults, 25 children) was not directed to any external object, the movements for adults in the positive field were 53.8 per cent., while those in the negative field were 46.2 per cent. For children the corresponding movements were 27.8 per cent., and 72.2 per cent. respectively. The two forms of experiment thus give about the same results. Combined they show the movements of adults in the positive field 52.5 per cent., and in the negative field 49.5 per cent., while for children in the same respective fields the movements are 27.3 per cent. and 72.7 per cent.

A comparison of movements in the right and left fields (reagents 74, cases 1,054) gives, for adults and children taken separately a result nearly alike. For both together the right hand moves in the left field 64 per cent., versus 36 per cent. to the right; while the left hand moves in the right field 66 per cent., versus 34 per cent. to the left. This means that normally the hand moves inwardly toward the median plane of the body.

As to the relative directness of movements it was seen that adults are much more direct than children. In adults 71 per cent. were fairly direct, and 25 per cent. were irregular. In children 21 per cent. were fairly direct, and 66 per cent. were very irregular. The changes of the original direction in adults were 32 per cent., in children 68 per cent.

Of right-handed persons, 75 movements were to the left, and 100 to the right. Of left-handed persons, 10 movements were to the left, and 15 to the right.

It was also found in adults 87.9 per cent. of the movements imitate the direction of an object followed in motion, while 12.1 do not. In children 81 per cent. imitate, while 19 per cent. do not.

With the eyes shut, while the object is moving, 98.2 per cent. moved in the direction of the object. Some even moved the body. Repetition increased the susceptibility. In all the experiments there does not seem to be any sex or age difference in children.

CHRISTISON.

42. THE FORCE AND RAPIDITY OF REACTION MOVEMENTS. Delabarre (*Psychological Review*, 4, 1897, p. 615).

The author made two series of experiments on fifteen subjects. A complicated and ingenious method was adopted, the subject being seated comfortably in a chair and told to react to a given signal. He

was instructed to think only of making a quick reaction, allowing the force to take care of itself, while his right forefinger and thumb were in touch with a pair of "jaws" connected by an electrical machine with a revolving drum and a column of mercury. With his eyes closed, the subject reacted to the sound of a signal key.

These experiments showed:

1. Dividing pressure index by the duration time, the resulting quotient representing the rapidity of contraction of the reacting muscles tends, for the same individual and the same series, to be constant.

2. There are well-marked differences between the different individuals and the two series of the same individual. They differ absolutely and in range of variation.

3. Although the two series overlap each other a great deal, yet in no case is the maximum value of the quotient of the pressure divided by duration greater in one series than in another.

4. The degrees of pressure exerted and the range of its variation are characteristic of the individual.

5. In every case but one the average pressure was greater in the second series, while the rapidity was greater in the first series.

CHRISTISON.

43. A STUDY IN THE PSYCHOLOGY OF RELIGIOUS PHENOMENA. J. Leuba (*American Journal of Psychology*, 7, 1896, p. 309.)

The fundamental resistance to conversion is self-assertion, and so self-surrender is the turning-point in conversion; that is, what we call *will* weakens as delivery approaches and resignation to God ensues. The land, so to speak, passes under a new rule and, henceforth, all strife ceases; harmony, a sense of unity and corresponding joy pervades the organism; everything becomes new and a new organic life begins. Pride is often the centre of the residual resistance. The diversity of feelings apparent in the conversion-experience of various persons are (1) desire for humility, (2) sense of impotency and unutterable woe, (3) prostrate in complete self-surrender, (4) confidence and expectancy, (5) love impulse to faith in God and Christ. The following are the usual stages in conversion: conviction of sin, humility, impotency, utter wretchedness, despair, self-surrender, hope, trust, love, faith. Leuba quotes Col. H. H. Hadley, for many years superintendent of the Jerry McCauley Water Street Mission, New York, in reference to conversions, and his own experience thirteen years ago. He says, "men have been converted in the delirium tremens. It knocks all the theology higher than a kite. I don't understand it, but it is so. Take my own case: A big, bloated drunkard—had fifty-three drinks the day before I was converted, most of them brandy cocktails, and before me I saw my Lord crucified." He had previously listened to the "experiences" of 25 or 30 converts.

CHRISTISON.

44. THE RELATION OF DIABETES TO INSANITY. C. Hubert Bond. (*Jour. of Ment. Science*, 42, 1896, p. 36).

The author studied 268 consecutive admissions of male patients, making an examination of urine in 175 cases and finding sugar in 12 cases. With these 12 cases he placed four found among the women, making 16 for analytical study.

His conclusions seem to be that, though usually quite rare among the insane, the proportion is increased by taking the recent cases. He finds no very clear relationship of the glycosuria to the form of insanity to the age or seemingly to the bodily health. Half of the cases had a history of alcoholic excesses. Two cases followed by autopsy had cirrhotic liver and kidneys.

45. ÜBER DIE VERÄNDERUNG DER PUPILLENREACTION BEI GEISTESKRANKEN (Concerning the Changes in the Reaction of the Pupils in Persons with Mental Diseases. E. Siemerling (Berliner klinische Wochenschrift, 33, 1896, p. 973).

A difference in the pupils may be found in normal persons. The Argyll-Robertson pupil is rightly regarded as an early sign of paresis. Persistent unilateral rigidity of the pupil is exceedingly rare. Siemerling has found in few cases of paresis the peculiar reaction of the pupil described by Gowers. The pupil contracted to light but soon resumed its former size, notwithstanding the illumination of the eye. The paradoxical reaction—dilatation of the pupil to light and contraction in darkness—is exceedingly rare. The difference in the size of the pupils, often associated with rigidity, may vary. This is especially true of paresis. Hippius has been observed in different forms of nervous disease.

Siemerling reports the condition of the pupils in 9,160 cases of mental disease observed at the Charité during ten years. Reflex rigidity was noted in 1,639 cases. The report is as follows:

Paralysis progressiva.....	1,524
Tabes with psychoses.....	29
Dementia senilis.....	19
Syphilis of the central nervous system.....	17
Focal lesions.....	19
Alcoholism	15
Injuries of the head.....	1
Epilepsy	4
Hysteria	4
Paranoia	7

Siemerling has found cerebral changes in dementia senilis. The pupils in the aged are narrow, and the reaction may be slow.

The explanation of pupillary rigidity, due to injuries of the head, is difficult, but the writer will not deny the existence of rigidity from trauma. The period of observation was short in the cases of epilepsy and hysteria, and rigidity as a sign of hysteria remains to be proven.

In two of the seven cases of paranoia there was a suspicion of tabes. The period of observation in the cases of paranoia was also short. The reflex rigidity may be the only sign of paresis for many years.

Siemerling has observed variations in the size of the pupils preceding or succeeding epileptic attacks. In one case rigidity of the pupils persisted several hours after the attack. The rigidity of the pupils in a convulsive attack is a diagnostic sign of great value in distinguishing between epilepsy and hysteria.

It is probable that centripetal pupillary fibres are to be found within the optic nerve, and that there is a partial decussation of these fibres.

SPILLER.

46. SYPHILIS AND PARALYTIC DEMENTIA IN ICELAND. Ehlers. Ugeskr. for Læger, I., 41.

The much-discussed relationship of syphilis and general paresis receives some new light from the author's studies from Iceland.

Syphilis is there a rare disease; the general idea of immunity is not, however, a correct one. The relative infrequency of the disease being related to the isolation of the various families at great distances from each other, rather than a result of any moral factor.

General paresis is unknown. The only case that the author was able to find having occurred in a sailor who had lived a number of years in foreign countries and had acquired syphilis there.

THERAPY.

47. DIE ERGEBNISSE DER LUMBAL PUNKTION (Lumbar Puncture). Fleischmann. (*Deutsche Zeitschr. für Nervenheilk.*, 10, 1897, p. 337).

The author reports on the lumbar puncture of 54 patients (according to the table 55) in the service of Lichtheim at Königsberg. It agrees with nearly all others that the procedure is without serious therapeutic value. Even in so-called serous meningitis only one of the four cases upon which it was practiced showed any good results from the operation. In accord with previous observers, however, is the conclusion that the technique is simple and facile, and the few unpleasant results of no serious import, and not to be regarded as a contraindication. Together with most other investigators of the subject, he lays most stress on the diagnostic importance of the abstracted fluid.

Puncture was done 15 times in 12 cases of tubercular meningitis, and the bacilli were found nine times in eight patients, while of five punctures in two cases of epidemic cerebro-spinal meningitis only one yielded the Weichselbaum coccus.

Four cases of purulent meningitis are recorded. Pus corpuscles and streptococci were found in the fluid of two, streptococci without pus in one, and many white blood corpuscles without micro-organisms in the fourth; that is, a positive finding, more or less conclusive, in all.

In studying this report, as indeed all others on this subject, the thought is inevitable that even when lumbar puncture is an undoubted diagnostic aid, the information thus obtained, considering the present status of therapeutics, is not of great practical value. As between tubercular meningitis and purulent meningitis, or even as between tubercular meningitis and serous meningitis, it must be acknowledged that a positive diagnosis is really of not very great value in directing the treatment or affecting the result of the disease.

Of the cases reported in detail, we may mention three of serous meningitis, as the disease is not very well known, and the cases illustrate some of the difficulties of diagnosis by means of lumbar puncture.

A young woman of 24 years was taken suddenly ill with violent headache, nausea and vomiting. There were soon added attacks of general convulsions, with loss of consciousness and moderate cervical pain. Seven days after the onset examination showed elevation of temperature, a dicrotic pulse of 44, pain on bending the head forward, and double optic neuritis. The following day the patient vomited several times and had a general convulsion lasting about two hours. Afterward she was quite rational and without fever. The next day there were removed by lumbar puncture 25 cubic centimetres of fluid, which contained one part per thousand of albumin, and in which a slight coagulum formed spontaneously. No immediate good effects of the puncture were discernible, but the patient gradually improved, and four weeks after the beginning of her illness was completely well, the persisting optic neuritis (which also rapidly improved) being the only sign of disease. The percentage of albumin in the fluid, as well as the spontaneous formation of coagulum, pointed to an inflammatory affection. As the patient belonged to a tuberculous family, and had herself suffered from scrofula and bone tuberculosis, tubercular meningitis was suspected, but examination of the fluid for tubercle bacilli was negative, and purulent meningitis was

excluded on account of the low percentage of albumin, the absence of pus corpuscles and micro-organisms. The rapid recovery of the patient was considered to verify the diagnosis of serous meningitis.

The second patient, a sailor aged 22, who had also had tuberculous osteitis, was taken with headache, nausea, vomiting, cervical rigidity and sleeplessness. The pulse was only 34. After a couple of weeks he rapidly improved, but four weeks after the beginning of the trouble the same symptoms returned, with a pulse of 48 and double optic neuritis. Three months later lumbar puncture was made and fluid removed which contained only 3-10 of one part of albumin per 1,000 and did not coagulate. After four weeks a second puncture drew fluid of the same character. There was no perceptible effect from the operation, but the patient improved, and was discharged cured four months from the first onset of his sickness. In this case the small amount of albumin in the fluid and its failure to show coagulation indicated a non-inflammatory affection, and yet the course and termination of the disease seemed to prove it a serous meningitis.

In the third case autopsy confirmed the diagnosis. A child of three years who had had eclampsia at 10 days suddenly became ill with high fever, headache, vomiting, loss of consciousness and rigidity of the entire body, but remained sick only a short time. Three weeks later she had a fit, with loss of consciousness, clonic spasm, followed by loss of speech and paralysis. On admission there were rigidity of the spine, impaired consciousness, rotatory movement of the head, continual grinding of teeth, slight paresis of right side and double optic neuritis. During the period of observation the pulse remained high, the temperature occasionally high but generally normal. The spinal canal was punctured twice, the fluid containing only a trace of albumin and developing no cloudiness. Three weeks after admission the child developed pneumonia, which was quickly fatal. The autopsy revealed internal hydrocephalus, spinal meningitis, catarrhal pneumonia and swelling of the internal follicles. This case, as well as others, goes to show that the serous meningitis of Quincke is probably not a perfect entity, but that approximately the same symptom-complex may be developed by a variety of conditions. It will also be noted that the quality of the fluid indicating inflammation, viz., large proportion of albumin and spontaneous coagulation were wanting, although distinct inflammation was present.

In another case, in which the diagnosis lay between tumor and abscess, the high pressure, equal to 45 millimetres of mercury, decided the observer in favor of tumor—a conclusion shown to be correct by operation and autopsy.

Another interesting case may be mentioned in brief. A boy of 8 became rapidly sick with all the principal symptoms of meningitis, but a few days later the condition seemed somewhat anomalous, and a lumbar puncture was made for diagnostic purposes. The fluid was clear, contained only a trace of albumin and showed no sign of cloudiness on standing; hence an inflammatory affection of the cerebro-spinal meninges was excluded. This being done, typhoid fever seemed the most probable disease, and the serum test being used gave a positive result—the correctness of which was fully confirmed by the subsequent course of the case, as well as by the diazo test of the urine.

PATRICK.

48. UN CASO DI GUARIGIONE DI MENINGITE CEREBRO-SPINALE DA DIPLOCOCCO DI FRAENKEL (A Cure of Cerebro-Spinal Meningitis). *Ibid*: Un 2 Caso di Guarigione, etc., Contributo ad valore diagnostico e Terapeutico della Puntura Lombare. A Second Cure, etc. Contribution to the Diagnostic and Therapeutic Value of Lumbar Puncture). Jemma (Riforma Med., 1896, pp. 259 and 260).

The author reports two cases of cures of cerebro-spinal meningitis, believing the feasibility of a certain diagnosis by means of lumbar puncture. From the first patient 20 cc. and from the second 40 and 35 cc. of a sero-purulent fluid, rich in pus corpuscles and diplococci, were withdrawn. He has always found the operation a safe one, and thinks it to have facilitated recovery in both of the above cases. In his second case attention is called to the favorable results obtained by the use of 15-minute hot (40°) baths. Judging from his cultures and experiments on animals, the author concludes that the cerebro-spinal fluid has the property of diminishing the virulence of the carriers of infection, and promises another communication on this subject.

VOGEL.

49. LA PONCTION LOMBE-SACRÉE (Lumbo-Sacral Puncture). Chi-pault (Journal de Médecine, 1897, p. 253).

Instead of making the puncture, as do most operators, between the third and fourth lumbar vertebræ, this author introduces the needle between the last lumbar vertebra and the sacrum, but his results have been very similar to those of other operators. He agrees with them that the operation is not difficult or dangerous, and is of decided advantage in diagnosis, particularly in the detection of tubercular meningitis. He goes so far as to assert that the examination of the cerebrospinal fluid thus obtained in cases of suspected tubercular meningitis is as imperative as the examination of sputum in cases of suspected pulmonary phthisis. Therapeutically, his results have been far from brilliant, but still we think better than those of most previous investigators. Of 19 cases operated upon, in 10 the result was absolutely negative; namely, 4 cases of infantile hydrocephalus, one probably caused by cerebellar tumor; one case of tubercular meningitis in the adult; 4 of general paralysis of the insane, and one of idiopathic epilepsy. In 4 cases the result of the operation was insignificant, namely, 3 cases of tubercular meningitis in infants and one of idiopathic epilepsy. In 5 cases the procedure accomplished more or less good. In one case of cerebral tumor in an infant three punctures at intervals of eight days relieved the headache and hebetude for a month and diminished the intensity of the choked disk. The fourth puncture in this case was without effect. In one case of congenital hydrocephalus the patient at the time of adolescence had begun to have attacks of severe headache with mental hebetude. At the time of the first puncture he had been suffering for 8 days and was relieved in 12 hours. Later attacks were aborted by puncture, and the patient was enabled to continue his occupation. In two cases of syphilitic meningitis with involvement of the cranial nerves, choked disk, mild delirium and attacks of stupor, a single puncture sufficed to cause a disappearance of the pressure symptoms and allowed time for active specific treatment which accomplished in one an entire cure and in the other almost a cure. One case of idiopathic epilepsy was relieved to a certain extent by punctures made every two weeks, the patient having during the 3½ months of treatment one attack a week instead of 8 or 10 in 24 hours. After cessation of the punctures he had 2 or 3 a week, which had been his usual number before the exacerbation for which puncture was done.

In conclusion the author states that in his opinion the future success of lumbo-sacral puncture must rest not upon the simple evacuation of the liquid, but upon its evacuation followed by the injection of an artificial serum which shall act either by the direct action of some contained medicament on the seat of the disease or by its inoculation effect analogous to that of anti-diphtheritic serum, and promises an early contribution to this method of treating infectious diseases of the cerebro-spinal meninges.

PATRICK.

50. LUMBAL PUNKTION (Lumbar Puncture). S. Bull (Norsk. Mag. for Lægevid, II, 1896, p. 498).

The author has performed lumbar puncture in four cases of tubercular meningitis. In one instance only 8 cc. were withdrawn and no tubercle bacilli found, while in the three other cases they were present, the quantity of fluid taken being 43-57 cc. Injection of this serum into the abdominal cavity of guinea pigs in one instance was, and in another was not, followed by results. Therapeutically considered, the puncture has a temporary palliative effect, but is of diagnostic importance. The author urges the necessity for caution, especially in private practice.

VOGEL.

51. SUL VALORE DIAGNOSTICO E CURATIVO DELLA PUNTURA LOM-BARE (The Diagnostic and Curative Value of Lumbar Puncture). G. Mya (Settimana Med., 1897, pp. 4 and 5).

G. Mya has performed 80 lumbar punctures in 23 cases, 15 of which were tubercular meningitis and encephalitis. Only in two instances were tubercle bacilli found in the fluid. His opinion is that when the serum is sterile and exudate-like (containing moderate numbers of leucocytes, flecks of ependyma, and clotting slightly) tuberculous is probably present, while in serous meningitis the fluid generally contains staphylococci, and in chronic cases is "transudate-like." He reports that in tubercular meningitis a distinct alleviation of the cerebral symptoms took place after the puncture. In two cases where clinically a diagnosis of the tubercular form had been made, the presence of staphylococci in the fluid pointed to an "early" meningitis, which the outcome showed to be the case. In cases of tumor the demonstration of a secondary hydrocephalus may be of greatest importance. Diagnostically, the author thinks highly of lumbar puncture; therapeutically, it may be of much value in some cases of acquired hydrocephalus.

JELLIFFE.

52. SUL VALORE DIAGNOSTICO E TERAPEUTICO DELLA PUNTURA LOM-BARE (The Diagnostic and Therapeutic Value of Lumbar Puncture). Jemina and Bruno (Estratto del Arch. Ital. di Clin. Med., 1896).

The authors give a very complete bibliography of the subject, including French, German and Italian works. Then follow reports on their own cases, 25 in number, as well as a thorough discussion of the operation itself and possible complications through the alteration of intracranial pressure. The physical, chemical, microscopical and bacteriological properties of the liquor are all of diagnostic importance. Clear or only slightly turbid fluid is found in tubercular meningitis; that of the infectious or epidemic forms is turbid or purulent. Of the chemical characteristics the most valuable is the percentage of albumen. In cases of tubercular meningitis it rises to 1 per cent.; in acute forms of meningitis it is higher than in chronic, and where brain tumors are

present may reach 3 per cent. and over. Of the greatest importance, however, is the bacteriological examination, and especially the proof of the presence of the tubercle bacillus, which, together with a clear fluid resembling the normal, was found in every one of four cases of tubercular meningitis under observation. The diagnostic value of the procedure is further upheld by the fact that negative results were invariably obtained in meningitis-like affections where some other disease was the source of trouble. Although the authors consider lumbar puncture an excellent palliative in many instances, its therapeutic usefulness is still to be determined. VOGEL.

53. DIAGNOSTISCHER UND THERAPEUTISCHER WERTH DER LUMBAL PUNKTION. DRUCKBESTIMMUNG MIT QUECKSILBER MANOMETER (Diagnostic and Therapeutic Value of Lumbar Puncture). Wilms *Münchener med. Wochenschrift*, 1897, No. 3).

Wilms presents the results of 30 lumbar punctures performed for various affections on 23 patients in the Augusta Hospital of Cologne. In epidemic cerebro-spinal meningitis the presence of the diplococcus intracellularis was demonstrated in three out of four cases, but tubercle bacilli were found only once in five cases of tubercular meningitis. The author prefers a Hg. manometer to the customary H_2O manometer on account of its compactness and readier manipulation. According to his observations the normal cerebro-spinal pressure averages 10 mm. Hg., while Quincke's figures are 40-60 mm. $H_2O=3.5$ mm. Hg., but many factors, such as the patient's posture, position of the head, outcries, breathing, etc., may influence the tension. The amount of fluid abstracted varies in different cases, and is not at all proportional to the pressure; 100 cc. were taken with good results from a case of cerebro-spinal meningitis, 25-60 cc. in cases of tubercular and epidemic meningitis; in non-inflammatory disorders the quantities range from 5-20 cc. VOGEL.

54. DIE DIAGNOSTISCHE BEDEUTUNG DER PUNKTION DES WIRBELKANALS (The Diagnostic Significance of Lumbar Puncture). F. Strauss (*Deutsches Archiv. f. Klin. Med.*, 57, 1896, p. 328).

In reporting the lumbar punctures performed in Ziemssen's clinic Strauss gives a detailed resumé of the present state of knowledge regarding the diagnostic and therapeutic significance of this operation. A noteworthy point is the fact that in cases of epidemic cerebro-spinal meningitis communication is frequently cut off between the cerebral and spinal sub-arachnoideal spaces. The bacteriological examination of fluid obtained from such cases revealed the diplococcus of pneumonia and the staphylococcus pyogenes albus and aureus. In the pathological cerebro-spinal fluid of a case of serous meningitis a chemical substance was found, which was either globulin or mucin. VOGEL.

Book Reviews.

HYPNOTISM AND ITS APPLICATION TO PRACTICAL MEDICINE. By Otto George Wetterstrand, M. D. Translated from the German edition by Henrick G. Petersen, M.D. Together with Medical Letters on Hypno-Suggestion, Etc., by Henrick G. Petersen, M.D. G. P. Putnam's Sons.

This little volume, which is dedicated to Dr. Liébeault, the founder of the Nancy School of Hypnotism, is not to be regarded as a treatise on hypnotic suggestion, for it consists, as its author states, "of unpretentious notes by a physician who, under the pressure of a fatiguing and engrossing practice, has not been able to develop his rich material into a more complete form;"—and the work should be judged accordingly.

The 117 pages of which the book consists are devoted very largely to the description of individual instances of disease in which hypnotic suggestion has been used for its therapeutic effects, together with comments on the results. The book is thus of an essentially practical character, and its value to practitioners depends mainly on the accuracy of the observations that are described, and on the possibility that similar therapeutic results can be obtained by other practitioners.

If it be true that the writer's experience is the record of cases observed with painstaking care and the insight which comes from broad clinical culture, this record of experience is surely of value to medical men. If, however, the author has approached his subject along a narrow path and with more enthusiasm than judgment, we can hardly be congratulated on having another book of a kind already too plentiful.

The basis of successful use of hypnotism for therapeutic purposes is necessarily the ability to render patients susceptible to suggestion. Wetterstrand has had a large measure of success in the induction of suggestible states, for of 3148 persons hypnotized only 97 failed to respond to his suggestion.

The author agrees with Liébeault, Bernheim and Forel that nearly every one is susceptible. He finds that age is an important factor in determining suggestibility, that persons under thirty are especially suggestible, and that all children from three or four to fifteen years of age are without exception hypnotizable. In the case of persons who cannot be rendered susceptible by the usual methods of hypnotizing Wetterstrand recommends the inhalation of chloroform, which was first used by Rifat of Salonika, for the purpose of overcoming such voluntary or involuntary resistance as the subject may offer, and of rendering him somnambulistic. There seems to be good evidence that the susceptibility to suggestion may in some instances be increased by the use of chloroform, but it is questionable whether the use of an anæsthetic for this purpose is likely ever to meet with the approval of representative English-speaking practitioners, however it may be regarded on the continent. In connection with the question of susceptibility it may not be out of place for the reviewer to give expression to the belief, based upon some experience with hypnotism that the average American citizen of American parentage, owing perhaps to greater curiosity and greater independence of thought and action, is considerably more difficult to hypnotize than the average continental citizen of corresponding social status.

While it is not within the scope of this review to present or discuss Wetterstrand views as to the numerous symptoms

and types of disease in which he has made use of hypnotism as a therapeutic agent, it is desirable to indicate the author's attitude toward representative forms of disease. We may select for this purpose some of the statements that are made with reference to insomnia, habitual headache, organic paralyses, hysteria, incontinence of urine, and external diseases. There can be no doubt that hypnotism is sometimes of great service in the production of sleep in nervous but healthy persons who are suffering from insomnia as the consequence of unusual nervous influences, such as worry, grief, etc. But it is surprising to hear that a woman who for seven years had been in the habit of lying awake for a large part of the night should be cured by an hypnotic séance with the aid of a few drops of chloroform. We do not wish to question the accuracy of this and similar observations, but think the author sometimes displays undue readiness to believe in the efficacy of his treatment,—for example as when he says, speaking of a case of insomnia cured by him: "I have not heard from him (the patient) since he returned home, but I have every reason to believe that his sleep continues satisfactory." In the section on habitual headaches a number of cases are described in which the head pains of neurotic individuals were greatly benefited. Here again the author seems to have been rather surprisingly successful. The reviewer is quite committed to the belief that most headaches ought to be treated by means directed against their cause, but that hypnotism should be tried where other means have failed, and that it occasionally is distinctly helpful in the relief of pain. The section on hysteria, though shorter than could be desired, is perhaps the best in the volume, in the sense that it displays the critical faculty in higher degree than it is elsewhere to be seen. In the pages on paralyses of organic nature we are startled by reading that an old hemiplegic who "could not lift the arm, which hung down powerless," was "completely cured almost after the first treatment!" "The paralysis had disappeared completely after six treatments and no difference could be discovered in the muscular strength of the left and the right arm." Neurologists are familiar with the spontaneous variations which occur from time to time in the power of hemiplegic patients, and there is some reason to think that suggestion may at times be capable of temporarily improving some paralyses, perhaps through altering the conditions of the circulation about the destructive lesion; but conservative men are not yet prepared to admit that a paralysis dependent on a lesion in the motor path can be even temporarily abolished by any form of psychic influence. In this particular instance we cannot but suspect that Wetterstrand either deceived himself in regard to the results of his treatment or erred in diagnosis.

In the section on incontinence of urine several instructive instances of improvement or cure are described as the result of suggestive therapeutics, together with some failures and relapses. The reviewer regards incontinence of urine in children as one of the conditions best adapted to the therapeutic use of hypnotism, and believes that practitioners will come in time to recognize the advantages of using suggestion for this purpose. In the author's short section on external diseases may be detected further indications of undue faith in the efficacy of suggestion directed against structural pathological conditions. A boy whose knee had been painful and swollen for about a month, as the result of a blow, limped into the author's office on October 13th, 1887. "The leg was in a semi-flexed position, and he (the patient) could not bend nor extend it. The joint was swollen considerably, with strong fluctuation." After hypnotization the patient could walk without limping; "all sensitiveness and pain had disappeared, and the joint could be bent and extended without any in-

convenience." On October 14th "the effusion had almost disappeared, but was still visible." It is within the limits of possibility that all this happened exactly as described, but the writer unfortunately makes the impression that he is unintentionally exaggerating his therapeutic results.

Enough has been said to show that the observations contained in this little volume are marred by indications of excessive credulity or loose observation on the part of the author. The histories of remarkable therapeutic results in organic cases would certainly have come nearer to convincing us of their accuracy had the clinical conditions been minutely and scientifically described. Although the looseness of description which characterises some of the histories detracts materially, in the reviewer's opinion, from the scientific value of the volume, it by no means destroys its interest. The book contains many observations which are probably truthful descriptions of the author's experience, and are well worth examination by those who are interested in determining the therapeutic value of hypnotism. It is just because the book contains so much that is interesting that we regret its shortcomings. The subject of hypnotism seems to attract especially persons of the artistic temperament, in whom the imagination often has undue sway. The writings of such persons, through their exaggerations, repel many sober-minded men, and thus the *facts* of hypnotism are being admitted more slowly by the medical public than might otherwise be the case. Krafft-Ebing in his recent "*Psychiatrischen Arbeiten*" shows how satisfactorily hypnotic observations may be presented by one who is a thoroughly trained observer. We still need in this field the mature and original judgment of men who have had long years of training in the methods of modern and scientific internal medicine.

The last quarter of the volume before us is devoted to medical letters on hypno-suggestion, etc., by the translator. These letters treat of hypnotic subjects in a general and rather diffuse manner, and can hardly be said to contain any information not already at our disposal. The section entitled, "*Music, not Sermons in Insane Hospitals*," elaborates a good idea, but unless the lyres of Orpheus are more tuneful than they usually are in public institutions, it may be questioned whether the frigid, grey-toned sermon is not the lesser of two evils. The translator's attitude toward thought transference and the starting up of "molecular action" in one brain by another is rather amusingly illustrated in the first letter. This does not detract from such general interest as his letters may possess, but it cannot fail to make us call in question, whether justly or unjustly, the accuracy of any personal views the writer may hold on the therapeutic value of hypnotism. The difficulty with the mental attitude of the man who firmly believes some things that have not been proved, is that he is at any moment liable to confound fact and fancy.

C. A. HERTER.

HYSTERIA AND CERTAIN ALLIED CONDITIONS, THEIR NATURE AND TREATMENT WITH SPECIAL REFERENCE TO THE APPLICATION OF THE REST CURE, MASSAGE, ELECTRICAL THERAPY, HYPNOTISM, ETC. By George J. Preston, M.D. P. Blakiston & Co., Philadelphia, Pa. 1897.

In this treatise of 298 pages, Dr. Preston has given a very full account of the protean manifestations of hysteria and of the various methods that have been suggested for the treatment of the disease.

The historical data collected in Chapter I., the review of the various theories regarding the aetiology and pathology of the disease given in Chapter II., are well calculated to give the special student such facts as he needs before taking up the further study of this interesting

subject. In the remainder of the book the author has avowedly kept the needs of the general practitioner in mind rather than those of the specialist, and yet every specialist who reads carefully will find facts, though familiar, carefully stated and described in clear and concise language.

It is evident that the author has given the subject a great deal of special study, so that the book is not merely a summary of the writings of others. That he has largely reproduced the statements and even the illustrations of the French school is excusable, and in as much as little that is new could be added to the exhaustive studies made by Charcot and his followers, the author has on the whole acted wisely in adopting this conservative policy.

In the chapter on Differential Diagnosis we find a useful summary in parallel columns of the characteristic symptoms of epileptic and hysterical "spells." The difference between neurasthenia and hysteria is brought out very clearly, but we think the author underestimates the difficulty of distinguishing between hypochondria in the female and hysteria. The former is a much more common affection than it is generally supposed to be.

It is a satisfaction to note that the author attaches very much more importance to the moral, hydrotherapeutic and general hygienic measures than to the treatment by drugs. His remarks on hypnotism are also to be commended for their brevity and sobriety. There are few books from which the general practitioner can get as readily the few salient points regarding hypnotism as he can from this monograph. We are in accord with the author in stating that the great value of hypnotism and the great service that it has done is that "it has taught us how to make our treatment of hysterical subjects suggestive." Furthermore, he is correct in stating that the successful treatment of hysteria consists not "in a suggestion now and then, as in the hypnotic state," but in continuous suggestion. It is far better to give the general practitioner this sober view of the matter than to praise indiscriminately the good effects of hypnotism and thus to encourage the practice of a questionable and not altogether harmless therapeutic method at the hands of the inexperienced.

Dr. Preston's book deserves to be read by the general practitioner who may be called upon to treat hysterical patients. But we commend it also to the specialist as a convenient work of reference on this perennially troublesome subject.

B. SACHS.

EYE-STRAIN IN HEALTH AND DISEASE. With Special Reference to the Amelioration or Cure of Chronic Nervous Derangements Without the Aid of Drugs. By Ambrose L. Ranney, A.M., M.D. Illustrated with 38 wood-cuts. The F. A. Davis Co., Publishers, 1914 and 1916 Cherry Street, Philadelphia; 117 West Forty-second Street, New York City; 9 Lakeside Building, Chicago.

Much of what the volume contains has already been published during the last ten years in various medical journals. The subject is one that has always proved of interest and about which many a wordy battle has been waged.

To many the claims made by Ranney for graduated tenotomy in the various conditions comprised under the collective term "heterophoria" will appear to be far-fetched, and the cures by him in chronic chorea, epilepsy, insomnia, and even insanity will seem to be no less than almost marvelous.

It is to be regretted that Dr. Ranney has not published the statistics of all of his heterophoric cases, including the failures as well as the cures. That he must have had many failures is evident when he

says that "one radical cure of epilepsy without the aid of drugs offsets a thousand failures as a scientific proof of a discovery." Such a statement may be considered to be extremely extravagant.

In spite of the adverse criticism which the methods of Ranney have evoked, we cannot help feeling that in view of the number of apparently severe cases, the cure of which is corroborated by different physicians, the subject deserves impartial investigation. The burden of such an investigation must, of course, fall upon the oculist, who, if not already entirely familiar with the technique of the examination and treatment as outlined in the book under consideration, could easily perfect himself therein.

It must be said that the claims of Ranney and of Stevens (his master on the treatment of eye-strain) were investigated some eight or nine years ago by the New York Neurological Society. The results of this investigation were not favorable to the authors of the new treatment. This, however, does not prove that there is nothing of value in the discovery and treatment of anomalous action of the muscles of the eyeball. Although it may seem incredible that the various heterophoric conditions should produce reflexly such grave disorders as epilepsy and chronic chorea, it must be borne in mind that errors of refraction may cause slighter nervous disturbances, as headaches; and, furthermore, that severe hysterical phenomena may be engendered by comparatively insignificant traumatism. The relation of eye-strain to intractable nervous affection is a subject which demands further investigation. As yet the data which we possess are too insufficient to permit us to draw definite conclusions.

MEIROWITZ.

CRIME AND CRIMINALS. By J. Sanderson Christison, M.D., Chicago. The W. T. Keener Co. 1897.

This little book of 117 pages is a reproduction of a series of articles contributed to the Chicago Tribune under the title of "Jail Types." The mode of origin explains the peculiar make-up of the book, and the occasional, careless and even incorrect use of language. (Vid. page 46, eighth line from bottom.) The author promises a larger systematic work on the same subject in the near future, so that the present volume may be taken to be a mere sketch of what he proposes to offer to the profession. The sketch has, however, some points of interest which it may be well to point out. The author divides the delinquents into three groups, viz., the insane (defective in reason); the moral paretic (defective in self-control); and the criminal proper (defective in conscience). Even the last-named delinquent is treated as the product of his ancestry and of his environment; and the doctrine of degeneration, although the author makes no reference to Lombroso or anyone else, receives full consideration.

We doubt the advisability of handling such a subject as this in an ultra-popular form. After all, the impression is created that there are extenuating circumstances in the commission of almost every crime, and just by so much, crime is made less abhorrent. One purpose of the author in writing these articles appears to have been to enlist the interest of good people in the question of prison reform; but the good people hardly need such incentives, and upon others not so inclined, such articles with their detailed narrative of crime, may have a very different effect. Moreover, the question of prison reform is one that calls for the exercise of sober judgment and mature deliberation, qualities which the ordinary newspaper reader does not possess to any serviceable degree. The author takes an advanced stand in advocating the abandonment of punishment as such by the State,

and favors the adoption of measures so as to turn out of prisons "better citizens than they receive."

The writer of "Crime and Criminals" is evidently an original thinker on the subject of which he treats, but we earnestly hope that he will hereafter appeal to the professional, rather than to the lay, public.

B. SACHS.

CLINICAL LECTURES ON MENTAL DISEASES. By T. S. Clouston, M.D., Edinburgh. Fourth Edition. Lea Bros. & Co. 1897.

There is probably no clinical treatise upon insanity in the English language which for clearness of statement surpasses this work of Clouston, which has now reached its fourth edition. It differs but slightly from the other editions, which are well known to the general practitioner and to the specialist, and, therefore, does not require any extended notice. The symptoms of insanity are presented in such a way as to remove much of the obscurity which seems to pervade the medical mind in regard to the clinical features of the disease, and the only criticism which we have to offer is that the more recent investigations in regard to the pathology of the disease are not adequately presented. The Bevan-Lewis methods of investigation are adopted, but there is very little notice of the revelations in the pathology of nerve cells due to the methods of Nissl, of Andriezen and of Berkeley. The illustrations are hardly of a character to commend themselves to the pathologist familiar with the appearance of lesions under the microscope, but in as much as this work claims to present the subject from the clinical standpoint, and in that respect is to be in every way commended, it is possible that these criticisms with regard to the pathological descriptions are not in place. For a student or a practitioner there is no work which can be more highly commended.

M. A. STARR.

BOOKS RECEIVED.

The Physical Correlation of Religious, Emotional and Sexual Desire. Jos. Weir, Jr., M.D. The Courier Jour. Job Printing Co., Louisville, Ky.

Rubiyát of Doc Sifers. By James Whitcomb Riley. Century Co., New York.

Hugh Wynne. 2 vols. S. Weir Mitchell, M.D., Philadelphia. Century Co., New York.

Eighth Annual Report of New York State Commission in Lunacy, Oct., 1895-Sept. 30th, 1896. Also Reprint of Second Annual Report.

Vol. xv., Transactions of the Iowa State Medical Society, 1897.

Vol. iv., Transactions of the Congress of American Physicians and Surgeons, held at Washington, D. C., May 4th, 5th, and 6th, 1897. Bulletin of the Ohio Hospital for Epileptics. Gallipolis, Ohio.

Twenty-fifth Annual Report of New York State Charities Aid Association.

The Aphasias and their Medico-legal Relations. By F. W. Langdon, M.D. The Lansing Printing Co., 1898.

The Psychology of Suggestion. By Boris Sidis, M.A. D. Appleton, 1898.

These Apresentada a Faculdade de Medicina e de Pharmacia. em 30 de Novembro de 1897. Por Julio Afranio Peixoto. Dissertação Epilepsia e Crime, Bahia, Brazil.

Festschrift anlässlich des fünfzigjährigen Bestehens der Provinzial-Irren-Anstalt zu Nietleben. T. C. W. Vogel, Leipzig.

THE
Journal
OF
Nervous and Mental Disease

Original Articles.

THE PSYCHICAL MECHANISM OF DELUSIONS.¹

By WILLIAM HIRSCH, M. D.,

Of all clinical symptoms of Psychiatry, there is none which even to the laity is so characteristic of mental disease as delusions and still it is more difficult to explain the psychical mechanism of this remarkable phenomenon than that of any other psychopathic condition.

Delusions have been recognized as such at all periods of history, and in fact it is this symptom which has given rise to the popular idea of craziness and madness. In spite of this fact, however, not even a satisfactory clinical definition of delusion has yet been offered. The layman is generally under the impression that the contents of certain ideas decide as to their delusive nature. We know, however, that while on the one hand the most absurd and incredible thoughts may emanate from an erroneous, illogical, but nevertheless, healthy state of mind, on the other hand an idea may correspond to real facts and still be a delusion. It is therefore much less the nature of the contents of an idea which leads us to diagnose a delusion than the way in which it manifests itself clinically by the actions and remarks of the individual.

The great difficulty of explaining the psychical mech-

¹ Read before the section on neurology on the New York Academy of Medicine, October 22, 1897.

anism of any psychopathic condition we will appreciate, if we consider that our knowledge of any normal psychical process consists only of hypotheses. All our modern psychological doctrines, ingenious and evident as they may appear, including the generally accepted association theory, are after all more or less speculations without absolute and irrefutable proof. One might even say, that it is idle work to try to explain the mechanism of a diseased condition as long as we do not possess the clear knowledge of its physiological analogue. However, the two sciences, psychology and psychiatry, form in more than one respect a mutual complement, and a thorough and accurate study of any psychopathic symptom is apt to throw additional light on the corresponding normal psychical process. In this way the study of psychiatric phenomena becomes of double importance to general science.

Ever since mental diseases have been made the subject of special study, one has tried to explain this interesting phenomenon—the origin and mechanism of delusions. Up to the present day, however, no satisfactory explanation has been given, a fact which is sufficiently well shown by the comparatively large number of theories which has been offered by various authors.

The old theory of a partial affection of the mind, which gave rise to the doctrine of monomanias, according to which the psychical condition of an individual could have been perfectly normal apart from a few isolated delusions, has been generally rejected. More thorough and careful observation has shown that the normal condition of the mind was seeming rather than real and that the delusions formed only a part of a general mental disease.

Another more recent theory sees a relation between the mechanism of delusions and imperative ideas. Both originate from a certain irritation of their anatomical location, and delusions often develop from imperative ideas, the only difference between the two being, that the form-

er are considered as real by the individual, thus influencing his whole psychical condition, while the latter appear as foreign and strange to the otherwise normal process of thinking, the patient himself trying to rid himself of this unpleasant disturbance.

This theory also has met with but little sympathy. In the first place it does not correspond to clinical facts to say that delusions ever develop from imperative ideas, and besides this the whole clinical course and aspect of these two classes of symptoms are so different from each other that we can not possibly assume the same or a similar mechanism for both phenomena.

A theory which has quite a number of adherents among modern psychiatrists, explains the origin of delusions by a primary disturbance in the process of association, by which the personality of the individual becomes changed. Parallel with the normal *ego*, a second morbid *ego* is formed, which latter gradually predominates and becomes responsible for the actions of the individual.

Apart from the metaphysical aspect which adheres to all these ideas of a metamorphosis of the soul or the *ego* and which is in fact nothing but a modernizing of antiquated metaphysical views, a theory based on a primary disassociation of thoughts could not by any means give a satisfactory explanation for the nature and origin of delusions. There exists in all probability a mechanism of compulsory associations, but such a condition does not produce delusions, but rather a certain class of imperative ideas.

A person, who in a compulsory way, associates the conception of a sharp pointed object with a certain unpleasant or painful sensation, may develop a symptom known as aichnophobia, but not a delusion. The condition of an individual, suffering from an hysterical psychosis, who is afraid to touch certain objects because there is a morbid association between these objects and the conception

"poison" is very different from the condition of a paranoiac who has a delusion of being poisoned.

Another attempt to explain the mechanism of delusions starts from the theory that every conception and idea is accompanied by a certain emotional state, which has been called the emotional tonus, and which, under normal conditions, stands in a certain proportion to the tonus of all other conceptions and ideas. It is this proportion of the intensity of the tonus of the different ideas to one another which according to this theory forms what we call the individual's character. The intensity of the tonus of those conceptions which form our ideas of honor, right, and wrong, etc.' determines our actions and modes of life. Under pathological conditions certain ideas may acquire an abnormally high tonus (*überwertige Ideen*), predominating over all other ideas, thus becoming delusions.

Although this theory is ingenious in some respects, it does not correspond to clinical facts. The author of this view (Wernicke), has himself carried his theory to its logical consequences, which finally led him to the assumption of isolated focal diseases of the mind. This theory bears a close resemblance to the old doctrine of monomanias.

Another theory, which has perhaps more supporters than any of the others, attributes the origin and nature of delusions to an intellectual weakness. Owing to the defects in his intellect the individual is not able to perceive and judge his impressions in the normal way. The lack of his full reasoning-power, leads him to misinterpret his environment and the actions of his friends. The important bearing upon general psychiatric conceptions, whether or not the presence of delusions necessarily involves a lack of intellectual power, and the strong opposition which this view has met with some psychiatrists, have caused some of the most prominent advocates of this theory, to attempt to prove that, in the affection which we might call *the* disease of delusions *kat' exochen*, i. e.,

chronic paranoia, there is always an impairment of the intellect, and that there is not a single paranoiac with a normal amount of reasoning-power.

It would take me too far to enter into a discussion of this latter question. What interests us here, would simply be what relation does the impairment of the intellect—if it exists at all—bear to the formation of delusions in cases of chronic paranoia? I think that everybody, no matter which view he holds regarding the condition of the intellectual power in paranoia must admit, that intellectual weakness alone is not sufficient to produce delusions. While we see on the one hand the highest degrees of weak-mindedness, imbecility and idiocy without any delusions, we find on the other hand among paranoiacs individuals with an intellectual capacity and reasoning-power far above the average. Jean Rousseau for instance, who said that the kings of Russia, England and France, all nobles, the women, the priests and mankind in general, had banded themselves together, and declared a dreadful war upon him, was a paranoiac, who suffered from distinct delusions of persecution, but still could anybody assert that his reasoning power, his intellectual capacity was too low to judge properly of contemporaneous events? Do we not find among our paranoical patients men whose logical train of thoughts and acuteness of mind, is decidedly above the average? There is one symptom, which can be frequently observed among paranoiacs especially in institutions, and which in itself involves a comparatively great amount of reasoning-power, i. e., dissimulation. A patient, for instance, who has delusions of persecution, knows that certain ideas are considered insane and that on their account he is kept in the institution. He learns how to hide these thoughts and pretends to have no enemies, etc. Is it logical now to assume, that it is lack of reasoning-power which produced these thoughts, while there is sufficient reasoning-power and self-control to hide them? If there is an intellectual impairment at all in

chronic paranoia, this can only be of a qualitative nature. The reasoning-power may be changed qualitatively, but as a rule is not diminished quantitatively. Apart from all these considerations, the so-called lowering of the ability to judge critically about the surroundings, even if we would admit its existence in paranoia, could only form one factor in the production of delusions, but would never be sufficient to explain the entire nature of this phenomenon.

The clinical aspect of delusions as well does not correspond to this theory. A weak intellectual capacity can be strengthened by training. Everybody knows that imbeciles and idiots can in some degree improve their intellectual capacity by the systematic training of their mental faculties. If, therefore, delusions were in any way due to an intellectual weakness, they ought to be influenced beneficially by training of the intellect, by advice and information. But just the reverse is the truth. While we may succeed to a certain extent in enlightening an imbecile person concerning his erroneous ideas, it is generally admitted that it is not only impossible to convince a paranoiac of the real nature of his delusions by logical arguments, but that such a measure would only be apt to aggravate the morbid condition of his mind. We are all in the habit as soon as the diagnosis paranoia is made to instruct the relatives never to try to argue logically with the patient about his delusions, yet this would surely benefit him, if his condition were in any way due to a weakness of his reasoning-power.

In close relation to this theory are all attempts to explain the formation of delusions in a purely psychological way comparing the mental process of paranoiacs with that of children or savages.

The principal reason why prominent psychiatrists hold so many different and directly opposite views concerning delusions, lies I think to a certain extent in the erroneous assumption that the delusion as such forms

a pathological entity, that the psychical mechanism of delusions must necessarily be the same, no matter of what nature they are and under what circumstances they occur. Up to the present time the starting point for any psychiatric investigation can only be clinical observation, and everybody knows how widely delusions differ clinically from one another in every respect. What right have we then to assume that one and the same mechanism lies at the bottom of all these different symptoms?

It cannot be denied that there are certain delusions, which can be explained by one or the other theory, mentioned above. The typical delusions of grandeur in general paresis is evidently due to a certain extent to a disturbance in the intellectual power. Corresponding to his pathological euphoria, the patient builds castles in the air, which his demented intellect is not able to correct and which are therefore taken as real. There is in these cases a direct proportion between the nature of the delusion and the impairment of the intellect. In the initial stages delusions of grandeur are confined to a certain amount of self-admiration. The more the dementia progresses the more they assume the true character of insanity. The businessman becomes a millionaire, the politician a great statesman, etc.

In other cases delusions might develop in a purely psychological way, to explain other psychopathic conditions. So for instance a person who is suffering from hallucinations may try to explain the voices or sensations, and thus construct ideas which bear the clinical character of delusions. In some cases of melancholia the self-accusation emanates from the great mental depression which makes everything, above all the *ego* appear dark and gloomy to the patient. In a similar manner, delusions, in acute mania, might be explained by a primary change of the moods and emotions.

There is, however, a large class of delusions, which might be called primary delusions or delusions proper,

which can not be satisfactorily explained by any of the theories offered. . Let us take for instance a person with a normal amount of intelligence, who suddenly refuses to eat, because without any apparent reason he suspects his nearest relatives to have poisoned his food. No persuasion, no argument, be they ever so clever and logical, are able to correct this error, to remove this delusion. Assuming that there are no hallucinations, no primary impairment of the emotions or affections, how can we explain the psychical mechanism of this condition?

Before I offer my theory for this condition, I would like to call attention to the very close relation which exists clinically between delusions and hallucinations. The contents of by far the greatest number of delusions as well as hallucinations refer mainly to the individual's own person. The figures and faces which are seen have either a threatening or an encouraging look. The voices may abuse the patient, call him names, threaten him or they may announce to him great revelations, tell him that he is the son of a crowned person or something similar. In an analogous way delusions have always reference to the *ego*. The occurrence of these two cases of symptoms, delusions and hallucinations, is also a very similar one. They occur in the same diseases under the same conditions. In chronic paranoia these two symptoms play an equally important role. According to the preponderance of the one or the other set of symptoms, we distinguish between paranoia simplex and paranoia hallucinatoria, although cases in which the one kind is missing completely are extremely rare, if they occur at all.

In view of this evident relation which these two phenomena present in their clinical aspect, it should appear but natural to assume that there exists a similar relation in their psychical mechanism. That the hallucination as such is not a clinical entity is a fact, which has been recognized and generally acknowledged for a long time.

Hallucinations may originate by a peripheral irritation, either in the peripheral organ itself or at any place of the sensory tract from the peripheral organ to the centre of perception. Such hallucinations are clinically characterized by a certain intellectual resistance and the fact of their being limited to one sense only. While these may be called primary hallucinations, there are others which arise secondarily to other psychopathic conditions, such as emotions and delusions. The well-known question of Griesinger, which he left unanswered himself: "Why does the patient believe in his hallucinations?" for instance in a voice announcing him to be the son of an emperor, can be answered for many cases: The patient does not believe himself to be the son of the emperor, because the voice told him so, but he heard the voice making this statement, because he believed it. The morbid idea, the delusion of grandeur was the primary affection, and the hallucination was secondarily produced. The realization of the fundamental difference between these various kinds of hallucinations caused some authors years ago to speak about contripetal and centrifugal hallucinations. Although at that time these terms were used in a more or less metaphorical way, they were based on perfectly correct clinical observation.

Modern anatomical investigation has placed the fact beyond any doubt that there exists only one nervous system and that all vital functions, no matter whether motor, sensory or psychical, are performed by the same kind of nervous material. It is therefore perfectly justifiable to assume, that all psychical disturbances are caused by the same disorders which we are accustomed to see in the peripheral part of the nervous system. Now which are the functional nervous disturbances known to us? There are in the first place the two principal symptoms, spasm and paralysis, which produce in the motor nerves convulsions, (tonic and clonic), and loss of motion; in the sensory system, hyperaesthesia, (pain) and anaesthesia. There is be-

sides a number of motor disturbances like tremor, ataxia, choreiform movements and athetosis, and in the sensory sphere we recognize many varieties of paraesthesia. If we apply these disturbances to the psychical sphere we will be able to explain many psychopathic symptoms. Paresis of the inhibitory apparatus will cause a condition of exhilaration as it is seen in maniacal conditions and in acute alcohol intoxication, while a spasm of the inhibitory apparatus might cause a retardation of association as in certain cases of melancholia. Psychical anaesthesia and hyperesthesia are well known symptoms. The involuntary compulsory movements of the choreiform or athetoid nature find their psychical analogue in certain morbid impulses, like coprolalia and similar phenomena which we are used to see especially in cases of psychical degeneration. Psychical ataxia might be called a certain disturbance in association as it is often seen in general paresis.

Besides all these disturbances there is a phenomenon to which I wish to call your special attention. Every practitioner is familiar with the different kinds of pain. Pain may be produced by a lesion at the peripheral end of the nerve, in the peripheral organ, or it may be produced by a disease of the nerve itself, as in genuine neuralgia or neuritis. There are other pains, however, of which hypochondriacal complaints form the most characteristic illustration, which originate in the psychical organ itself. How the patient explains these sensations to himself, whether he thinks that his organs are destroyed by a fatal disease or whether he attributes these sensations to external influences like telepathy, etc., makes no difference as to the mechanism of the phenomenon. We have to deal in all these cases with sensory hallucinations. According to the theory generally offered for conditions of this kind there is some central irritation, which by force of habit is assigned to the peripheral organ. This theory is open to a good many objections, so that it might be necessary in order to explain fully these phenomena to assume a re-

verse, i. e., a centrifugal instead of a centripetal action of the sensory tract.² Be this as it may, the fact is that there are cases in which the normal relations are reversed, in which central sensations produce the conception of peripheral irritation instead of being produced by them. That such a condition also takes place in the psychical sphere in the production of hallucinations I have previously mentioned. Now let us go one step further and consider primary delusions from this point of view.

The normal psychical process starts from the simple sensation of the different organs of sense. Sensations combine with other sensations and form a perception. Through the combination of perceptions and the action of apperception originate conceptions, which by the process of association form complicated thoughts and ideas. Every combined conception is followed by a certain emotional state, generally called mood, which persists until it is replaced by another emotional state. These states survive their underlying conceptions, their duration standing in a certain relation to their intensity. In cases of high tension they may influence the emotional states of the following conceptions, shading them with their own colors. The complicated ideas and thoughts with their emotional states thus form the last station of the centripetal psychical process, and at the same time are the starting point of centrifugal or psychomotor actions. If we analyse the psychical process into its components, we find as the fundamental elements the sensations of sense, from which in a centripetal way the process starts in the following order: Sensations of sense—perceptions—conceptions—thoughts and ideas (conclusions)—emotions and moods.

² Shortly after this paper was read I noticed an article by Benedikt (*Die doppelseitige Leitung der Nerven. Deutsche medicinische Wochenschrift*, 1897, No. 41), in which he tries to explain hypochondriacal and hysterical pains by a centrifugal action of the sensory nerves.

Now let us apply the phenomenon which we have observed in a part of the sensory tract, i. e., the retroaction, to the whole psychosensory sphere. The ultimate link in the great chain of the process of thinking, the conclusion, the ready formed idea, either produced by some emotional state or some other internal cause, such as fancy, dreams, etc., forms the origin of the psychical mechanism. In a centripetal way it is analyzed into its components, is transformed into various conceptions, which may go on to produce real perceptions, i. e., hallucinations. We would have the same mechanism for the delusion as we have for certain hallucinations, and I think, it would fully correspond to clinical facts to call a delusion a hallucinatory idea. *The morbid condition does not rest with the formation of the idea, or perhaps a primary emotional state as such—*for these may occur under perfectly normal conditions—*it lies in this retroactive mechanism, by which the baseless conclusions take the character of reality, just as endogenic perceptions are transformed into real images, into hallucinations.*

Let us now from this point of view look at our cases of chronic paranoia. In the first place we now understand the clinical relation between delusions and hallucinations. We see why under certain conditions, i. e., in cases of primary delusions, the patient believes unreservedly in his hallucination, while under other circumstances, i. e., in centripetal hallucinations he himself considers his hallucinations as morbid symptoms and only gradually, because of their persistence, begins to consider them as true. We see further, why delusions always bear close reference to the individual's own person. For it is clear that the contents of ideas and thoughts which are not formed in the usual way by observation and conclusions, but which come from within, which form not the end, but the starting-point in the process of thinking, necessarily must possess the character of their source. We can also account now for the

characteristic peculiarity of delusions, not only to withstand all logical arguments, but to be aggravated by them instead of being corrected. It is clear that any ideas which have not originated by logical conclusions from a consecutive chain of thoughts, cannot be corrected by logical arguments. The physiological error is caused by illogical conclusions or incorrect observation, and therefore will readily yield to correction. But an idea which comes from within can not be influenced from without. To try to correct delusions by arguments must therefore necessarily be just as fruitless as to try to make the water in a river run towards its source.

It will be now of special interest to consider the general mental condition of paranoiacs from the standpoint of the theory of retroaction. In some cases of acute paranoia, where the retroaction takes place more or less in the whole psychical organ, we see a flood of delusions break over the patient. All surrounding events are interpreted; normal perceptions and conclusions seem entirely impossible. If the retroaction penetrates down to the centres of perception, we have the hallucinatory form of paranoia. The surroundings may then not be perceived at all or only in a fragmentary way. The patient's attention appears to be entirely absorbed by his hallucinations and he seems to live in another world.

By far more interesting are those chronic cases of paranoia with more or less isolated delusions and hallucinations. As mentioned already, the general mental condition, especially the intellectual power of paranoiacs has of late years been made the subject of numerous discussions and controversies. While there are on the one hand advocates of the view that a person can be perfectly normal apart from one or a few isolated delusions, there are on the other hand authors who think that in every case of paranoia there exists a marked diminution of the intellectual power. The former view is evidently due to

insufficient observation. There is not one paranoiac whose mental condition beyond his delusions can be called normal. Delusions as such form only some of the clinical manifestations of the morbid process of thinking, and if we could take away the delusions, the person would remain just as insane as he was before. As to the latter view, the affection of the intellect, I have expressed my opinion before. The reason that even excellent observers and men of vast experience believe, that a certain amount of intellectual weakness forms an essential part of every case of paranoia, lies to a certain extent, in the great contrast which the contents of delusions frequently show to conclusions formed in the normal way, and in the inability of the patient to realize the morbid nature of his delusions. We cannot fail to understand the queer actions and the peculiar mental condition of a paranoiac, if we bear in mind the effect which a retroactive psychical process must necessarily have on his whole psychical condition. A baseless idea, a judgment without logical foundation forms the starting-point of a long chain of thought and may cause the individual to draw from it all sorts of conclusions which, as a matter of course, are apt to influence the moods and emotions, which in their turn might again become the source of new retroactive processes. But the strange contents of delusions must by no means be considered as evidence of a weak intellect. All sorts of ideas, even highly ingenious-looking thoughts, might be produced in this retroactive manner, a fact which is sufficiently shown by the large number of paranoiacal prophets, poets and artists in history. This theory of retroaction may therefore furnish, perhaps, another clew to the alleged relation between genius and insanity, although, as I have always held, a true genius is just as little insane as a paranoiac is weak-minded. We are not more justified to conclude weak-mindedness from the contents of delusion than from a very silly dream, which as everybody knows, might be experienced by the most intellectual

people. Both phenomena stand entirely out of reach of the intellectual reasoning power.

The fact that this disturbance in the psychical mechanism in most cases affects the process of thinking only to a certain extent, but leaves a considerable amount of normal thinking and logical conclusions undisturbed, is in perfect harmony with the analogous process in hallucinations. A person may suffer from hallucinations in one or the other organs of sense, but at the same time have perfectly clear and undisturbed perceptions.

The fact that emotions often form the starting-point of primary delusions, furnishes an important indication as to the treatment. As long as we are unable to exert any direct influence on the functional disorder itself, i. e., on the nervous retroaction, our efforts must be directed towards the possible source, the emotion. Every one who is obliged to deal with paranoiacs knows by experience that the best way to get along with these unfortunate patients consists in a careful avoidance of all exciting agents, may they come from within or without, and that these patients will do best if their mind is absorbed by some mechanical occupation, which will guard the moods from injurious fluctuation. The beneficial result of this purely empirical treatment furnishes therefore a further support to the theory of nervous retroaction.

The attempt to explain all psychopathic conditions by disturbances known to us in the so-called peripheral nervous system, stands in full harmony with modern anatomical research. Modern investigations have shown that the mysterious psychical organ is made up of precisely the same material as the peripheral part of the nervous system. There is no difference between an intracerebral and a cerebrospinal or spino-muscular neuron, and what holds good for the one must hold good for the other. In the same manner as the anatomical difference between the central and the peripheral nervous system has disappeared, we must learn to classify all disturbances of the entire nervous system on a uniform base.

Only if we succeed in explaining all psychopathic symptoms by a strictly physical mechanism, if we succeed in freeing ourselves from all metaphysical notions, the bridge between neurology and psychiatry will be completed, and the study of mental diseases will cease to stand apart from the other branches of clinical medicine.

EDUCATIONAL USES OF HYPNOTISM.

R. Osgood Mason, M.D., (Pediatrics, Feb. 1st, 1897) reports the following cases. A girl of fifteen was intelligent, but had no aptitude for routine school duties. If she learned a lesson, it was forgotten in class room. Private teachers were employed to prepare her for an examination, but after months of efforts they reported it was useless for her to go on. At this time she was treated by hypnotic suggestion. Improvement was immediate, both in ability to study and recite. After six treatments she greatly surprised her teachers by passing her examination with a percentage of 79, which entitled her to come up for a college examination, and later on passed her entrance examination with a percentage of 88. An intelligent but uneducated woman, although a good reader, experienced great difficulty in spelling. All her life she had been a sleep walker. She was an excellent hypnotic subject, and a single treatment entirely cured her somnambulism, so that she has not left her bed for two years. Hypnotism was also tried for her inability to spell. The effect was immediate, and after two or three treatments she wrote a four-page note without consulting a dictionary—with only two or three errors. Her language was that of an uneducated person, but after half a dozen suggestions it became greatly improved, though not faultless. A little boy was a most unhappy coward, afraid of pain and a cry-baby among his playmates. Hypnotic treatment caused a marked change in his manner—all crying and cowardice disappeared and he is now self-reliant and happy. A little girl was troubled with night terror. She slept soundly when first put to bed, but after two or three hours awoke screaming on account of a hideous black man she saw in her dream. Her sleep, since under hypnotic treatment, has been perfect. A boy, addicted to self-abuse and cigarette smoking, had a poor memory, was backward in studies, dejected and unmanly. The habit of self-abuse was cured in one month, and he finally only smoked one cigarette a week. There was also great improvement in his memory and interest in studies. A young man suffered from morbid sexual ideas and practices of the homo-sexual type. A week after treatment he reported almost entire freedom from his troublesome instincts and imaginings. A young man whose dominant idea had reference to disease, feared and expected to be attacked by every ailment which he heard of. He was unable to attend to business, and had frequent suicidal impulses. He was a good hypnotic subject and the cure was complete. Dr. Mason remarks that, in none of these cases has the patient's will been weakened—in no case has he been made dependent upon the hypnotizer, nor has any hypnotic habit been formed; the power of self-control has not been diminished, but, on the contrary, he has been helped to do the very thing which in his best moments he desired to do and of himself was not able to accomplish.

FREEMAN.

EQUILIBRATION AND ITS RELATION TO VERTIGO.¹

By FRANK K. HALLOCK,

Cromwell, Conn.

The following physiological considerations may be regarded as preliminary to a future discussion of the origin and mode of development of the various forms of vertigo. The chief point of this article is an attempt to show the importance of the relation of the cortical centres to the act of equilibration.

Vertigo is essentially a psychical phenomenon and may be defined as the consciousness of a disturbance of the body equilibrium. The maintenance of equilibrium represents the action of those forces in the organism having to do with the preservation of the position of the body in space. This preservation of the body equipoise is only one of the acts comprising the general process of equilibration which consists of the balancing of all forces operating in the organism. These forces are called functions, and equilibration, therefore, in its broad philosophical significance is the maintenance of the physiological balance. Frequently, however, the term "equilibration" is used in a restricted sense as meaning simply the act of maintaining the equilibrium. Thus limited and defined, the expression will be more convenient, and for the present purpose will be so employed.

Under normal conditions the act of maintaining the equilibrium is carried on automatically and independently of consciousness, and it is only when there occurs a defect in the mechanism of the process that we become conscious of the result of its defective operation. This result is that peculiar sensation called giddiness or dizziness, or when

¹ Presented at the Annual Meeting of the American Neurological Association, May, 1897.

the sensation is more intense, it is termed vertigo. At no time are we conscious of the process of equilibration either in its defective or normal action. We are conscious only of the results of this action, which in the case of defective equilibration is the sensation of vertigo, and in the case of normal equilibration is the sensation of maintained equilibrium. Ordinarily, this latter sensation is not perceived in consciousness unless it is made the object of special attention. When this is done the act of equilibration ceases to be automatic and becomes in part voluntary, that is, it is associated with consciousness and, therefore, may be said to have a psychical element. The simplest kind of action which will illustrate the operation of this psychical element is that of voluntary body balancing. In such effort the action of muscle in correspondence to sensory stimuli does apparently yield distinct sensations which can best be described as those of lost or maintained balance. It is also possible to conceive of these acquired reflex sensations as of frequent occurrence in the consciousness of a child learning to walk. As the motor adjustment to the sensory stimuli becomes more perfect, the resulting sensation grows less vivid in consciousness until, finally, by constant repetition, the act of equilibration, like the act of coordination, with which it is closely associated, becomes purely automatic. The term automatic, rather than reflex, is used to denote that the act is a complex of reflex acts and may be subject to modification by intercurrent cortical influence.

In this act of body balancing, which represents the mildest form of equilibratory disturbance, there is no true vertiginous sensation. Such sensation as does exist is the result of a series of sensori-motor processes, each short, complete and successful in its alternating loss and recovery of the body equipoise. If we were to imagine a rapid succession of ineffectual attempts to regain the body balance, then the sensation of vertigo would arise in consciousness.

The act of maintaining the body equilibrium or equilibration, using the term in its restricted sense, as an organic process is dependent not upon the operation of a special equilibratory apparatus or centre, such as has been suggested to exist in the semi-circular canals and cerebellum, but upon the operation of all parts of the general sensory-motor system which in any way subserve the process in question. The action of these parts is not separate and distinct, it is combined, and yet the impairment or absence of one or more parts does not necessarily destroy the general function, it simply limits its extent and power. Thus, admitting the importance of the part played by the semi-circular canals, it is noted that their destruction does not apparently interfere with the continuance of satisfactory equilibration.

Likewise, loss of vision and impairment of the tactile sense may occur, and yet the body equipoise be maintained. Indeed, it is possible to conceive that all sensory stimuli may be diminished to the point of barely permitting the simplest motor co-ordinations, and still consciousness be not aware of disturbance of the equilibrium. Experiments with animals have shown that the function of equilibration may still persist after the removal of the cerebral hemispheres. This fact clearly demonstrates that consciousness is not an essential factor in the act. It is also certain that the cutting off of all sensation would render the phenomenon of vertigo impossible. The severest attacks of vertigo occur only when the sensory paths are in a condition to transmit powerful, but perverted stimulations to encephalic centres.

While there need not be, therefore, and commonly is not, a psychical element in equilibration, it is nevertheless true that in man this act in its very highest development may be considered not a purely physiological but a psycho-physiological affair. And viewed as automatic, it may be said to be by a species of retrogressive development the

result in part of conscious or voluntary acts. Thus, in the child learning to walk, or in feats of body balancing, the acts become automatic by practice, *i. e.*, by repetition, and what are largely voluntary acts, executed at first with the aid of consciousness, gradually lose their psychical concomitant and become automatic.

In the act of locomotion, which is an allied sensorimotor mechanism to that of equilibration, the psychical element is present to a greater degree. Yet it is constant experience that the act of locomotion once thoroughly inaugurated is frequently carried on apparently automatically, that is independently of consciousness for short periods at least. Equilibration is not only associated with locomotion, it also underlies it as a primary function. Locomotion cannot occur without equilibration, and on the other hand locomotion is not essential to equilibration, as it is conceivable for an individual to maintain the balance without being able to walk. The fitness and advantage of equilibration becoming automatic is obvious from the fact that no act of such constant and fundamental importance could have a psychical concomitant without seriously embarrassing the efficiency of action. In other words, conscious effort to adjust the body after each change of position would subject all movements to delay and imperfect execution.

Considering now the purely physiological side of equilibration, we find that the act consists of the operation of three sets of factors.

First, peripheral end organs with their afferent nerves conducting sensory stimuli.

Second, co-ordinating centres receiving these stimuli.

Third, efferent nerves from these centres conducting motor impulses to the skeletal muscles.

The first group of factors represents sensory stimuli arising from all sources in the periphery which are capable of yielding directly or indirectly sensations of the positions

of the body. The three chief sources of these stimuli are, first, organs and nerves in the skin receiving and transmitting tactile impressions. Also grouped with this class are the sensory nerves of the muscles, tendons, ligaments and joints which transmit impressions indicating the position of the limbs or body as a whole. This is the so-called muscular sense.

Second, the eyes with their retinal expansion receiving and transmitting visual impressions through the optic nerve.

Third, the semicircular canals of the internal ear receiving and transmitting through the vestibular branch of the auditory nerve stimuli, which indicate the position and balance of the head, and less directly play a part in the precision of movement and general state of equilibrium throughout the body.

The coordinating centres, located chiefly in the mesencephalon and cerebellum, are the second factor in the act of equilibration. Animal experimentation and pathological conditions in man show the connection of those centres both with the cortex above and the lower centres in the spinal cord. On one hand, following injury or absence of the hemispheres, the subcortical centres are seen to function satisfactorily for all simple peripherally initiated acts of equilibration, while the complex or originating acts fail of execution. On the other hand, injury to the mesencephalic or cerebellar centres causes direct and more or less permanent impairment of the simple as well as complicated acts of equilibration.

The third factor is the system of efferent nerves from these centres carrying motor impulses which excite muscular action, thereby adjusting the position of the body in accordance with sensory stimuli previously received.

The imperfect operation of any part or the whole of this triple mechanism may give rise to a vertiginous sensation which will correspond to the degree and extent of

the disturbance of this mechanism. In the order of importance the coordinating centres take precedence over the other factors, and it may be stated that unless the disturbance in any part of the mechanism also involves these centres no vertigo will result. They preside, as it were, over the functions of equilibration, and on their integrity depends the success of adjusting the efferent motor impulses to the afferent stimulations. These centres are subject to two classes of impulses, first, the peripheral afferent stimuli, and secondly, influences from the higher cortical centres, which, may modify or interrupt their action. In the case of absence or perversion of stimuli from any point in the periphery, *e. g.*, the eye, we note that the coordinating centre accommodates itself to the deficiency and carries on the mechanism for all ordinary acts of equilibration nearly as completely as before. The cortical or psychical influence on the equilibrical centres is seen under emotional conditions.

The efferent nerves are the least important factor in the general mechanism of equilibration. They, and the muscles they innervate, may be subject to serious disorder, and yet no vertigo result unless the coordinating centres from which the impulses are derived are likewise disturbed. This is seen in advanced ataxia. The movements of the legs may be so imperfect and uncertain that the patient cannot stand or walk, and yet no vertigo results because the sensation of lost balance ceases when the effort stops. The sensation of disturbed, or lost, balance must persist in consciousness in order to produce vertigo.

Pursuing in brief detail the study of the sensory stimuli on which the integrity of the equilibrical centres in the main depends, the truth of the assertion that the act of equilibration is of compound and not simple nature, is soon made evident. Considering first the influence of tactile and muscular sense impressions, it is noted that removal of the skin of the hind limbs of the frog, or Heyd's

experiment in man of benumbing the cutaneous nerve endings in the soles of the feet by chloroform, results in difficulty in standing and keeping the body balance. In locomotor ataxia we have the combined effect of impaired tactile sensibility and also disturbed joint-muscular impressions resulting from the ataxic condition. In maintaining the equilibrium the ataxia is relatively of less importance than the loss of skin sensibility. That is, the ataxia may be pronounced without marked equilibrial disturbance, and *vice versa*, the equilibrium may be greatly disturbed without ataxia. With the loss of tactile sensibility, say of the soles of the feet, however, there always occurs difficulty in balancing the body, and the amount of this difficulty seems to depend more upon the degree of impaired tactile sensibility than upon the degree of ataxia.

The effect of visual stimuli upon the above phenomena is very distinct, and shows the intimate relation existing between the tactile and muscular sense. In every instance the difficulty of maintaining the balance is immensely lessened provided the eyes remain open. Within certain limitations the impairment of both the tactile and muscular sense may be compensated for by the visual sense. The simple standing erect of the ataxic individual with eyes alternately open and shut will illustrate the close association of the two kinds of stimuli in their effect upon the equilibratory centres. In the blind the absence of visual impressions requires the higher development of the tactile and muscular senses, with a consequent much greater dependence upon stimuli of this character. But in the normal individual the two sets of stimuli are developed together, the eyes noting the point of contact and following the movement of the limbs in the various positions assumed independently, or in relation to external objects.

Visual stimuli may exert a disturbing effect upon equi-

libration in two ways. First, by the unusual movement and relation of objects in the field of vision, such as is experienced, on moving trains, or in regarding those passing, in watching swiftly running water, in looking over a precipice, etc. The visual impressions arising from the extraordinary relation of external objects demand new and uncommon efferent impulses which the equilibrial centres are not capable of furnishing. Hence, a failure in the motor adjustment to the afferent stimulation results, and consequent upon this failure there arise in consciousness feelings of insecurity, of abnormal body position and space relationship, of imperfect balance and dizziness.

Secondly, these same sensations may be experienced following perverted visual impressions dependent upon the defective operation of the oculomotor mechanism. The condition of nystagmus, or of paralysis of the external rectus muscle of the eyeball, yields disturbed visual impressions which produce the feelings in consciousness mentioned above.

Passing now to the consideration of the semicircular canals we come to the most important factor in the maintenance of equilibrium. Without attempting to define the exact nature of the semicircular canal stimuli, it will be sufficient to say that there is no reasonable doubt among physiologists that such stimuli exist, and that they play a part in the movements of the head and body, particularly in regard to the precision and equilibration of the muscular acts. The evidence of this function is derived through experimentation upon the canals in animals, and the pathological and functional disturbances of the ear in man. The work of Flourens and subsequent investigators is sufficiently convincing, and the disturbance of the equilibrium associated with affections of the ear in the symptom-complex, called Ménière's disease, is unmistakable proof of disordered semicircular canal stimuli. It has been argued

that these canals can not be such an important factor in equilibration as claimed, because we are so unconscious of their operation. It is true that the stimuli arising in the canals and carried by the vestibular nerve do not excite sensations such as result correspondingly to the visual or tactile stimuli, but this very fact may be said to indicate the deeper and vital nerve function of these organs. The acts of respiration, circulation and digestion under normal conditions yield no sensation. Similarly if we consider what a fundamental and vital process the act of equilibration is, how it lies at the very bottom of organic stability and the preservation of the body in space, then it is easy to see how the function should be independent of consciousness. It is a function, too, which has existed from the moment of birth, and, viewed psychologically, must have been present in an incipient degree previous to the development and incorporation of its visual and tactile factors. In considering the cutaneous sensations, it is possible to conceive that the atmosphere may produce by pressure upon the tactile endorgans continuous inflowing stimuli, which by reason of their constant and familiar existence fail to develop sensation. If a change in this pressure occurs beyond the ordinary limits, then the stimulus yields a sensation. In a similar manner one can conceive of the normal unconscious influx of stimuli from the semicircular canals. The ordinary atmospheric pressure without in conjunction with a given state of the endolymph of the canals—which is also subject to internal variations from moment to moment, owing to the position and movements of the head—may produce stimuli which affect the coordinating centres below the level of consciousness. If now the external pressure varies, being markedly increased or decreased, as occurs by forcing air in the ear, or in a caisson, or if the internal condition of the canal contents departs from the normal, *e. g.*, by sharp change in the tension due to circulatory disorder, then

a variation occurs in the character of the canal stimuli. There is this difference, however, between the change of stimuli of the skin and canals. In the former we are directly conscious of the change, as interpreted by corresponding sensations, *viz.*, touch or pressure. In the latter instance, by virtue of the change in stimuli, we are conscious of sensation corresponding to the stimuli, but of a totally different sensation, *viz.*, dizziness which cannot be traced directly to the canal stimuli.

This dizziness may be compared in character to the sensations of hunger, thirst, and nausea, which cannot be directly connected in consciousness with the stimuli originating them. All of these sensations are complex in nature, and there does not exist the simple immediate relation between the stimulus and sensation as in the case of pressure on the skin. Although of such a general and indefinite character, they are all more or less referable to that region of the body from which the stimuli come. Thus, thirst is referred to the mouth and throat, hunger and nausea to the stomach. The diminution of water in the cells of the mucous membrane of the mouth, tongue and pharynx may generate stimuli which are the chief factor in the production of a general body condition which is represented in consciousness as the feeling of thirst. In like manner, hunger and nausea may arise from the alterations in the gastric mucous membrane. In dizziness, especially of pronounced degree, aural symptoms are almost always present, but the condition is so general, involving, as it does, both visual and tactile factors, that the individual fails to refer it to the ear.

While the semicircular canals are undoubtedly a factor of most unique and special character in the act of equilibration, it is still true that their absence or destruction does not prevent the maintenance of the equilibrium. Animals deprived of their canals show marked impairment in their equilibrating power which is never fully recovered.

In deaf mutes and persons who have suffered from destructive lesions of the labyrinth, vertigo is a rare symptom, and cannot be induced experimentally with anything like the frequency that it can in normal individuals. The loss of the semicircular canal stimuli is compensated for in great part by the visual and tactile stimuli, but the complex and highly developed acts of equilibration are not possible, and the capacity of the individual to experience vertigo is correspondingly decreased.

As stated at the outset, under ordinary conditions equilibration is an automatic process, but it has been shown that whenever it is associated with conscious effort, it necessarily acquires a psychical element. That it is possible to introduce this psychical or subjective element at all times becomes apparent when we consider that the afferent sensory stimuli necessary to the act of equilibration are the same as those which yield the sensation of position in consciousness, that is, we are conscious from moment to moment of our position in space, and the relation of the body to external objects. Indeed, it may be said that equilibration depends for its existence on the combined action of stimuli which occasion sensations of position and sensation of motion, but we do not commonly make any conscious application of these sensations in the execution of the act. These afferent stimuli, therefore, may be said to have a two-fold effect, one is the production of pure sensations of position, and the other is the concomitant exciting of centres below consciousness, whereby the body equilibrium continues to be preserved. It is probably, as suggested in the early life of the child, that the sensations of position were made use of as a psychical factor in the act of equilibration, but gradually, as the act became more perfect, *i. e.*, more automatic, these sensations ceased to be necessary as being consciously related to the act. If the maintenance of the equilibrium can be defined as the action of those forces in

the organism having to do with the preservation of the position of the body in space, then the consciousness of a disturbance of equilibrium, or vertigo, may be interpreted as the consciousness of a disturbance of the body position. This definition of vertigo is, indeed, correct with the qualification that the disturbance is of a peculiar kind. It is not every disturbance or change of the body position which is temporary and due to inadequate motor acts, but rather a permanent disturbance both of the cortical and subcortical nerve centres, due to the continuous influx of disordered sensory stimuli from the periphery. The individual, therefore, becomes doubly conscious of disturbed spacial relation, primarily, as the direct result of the distorted sensory stimuli on the cortex, and secondarily, as the result of the disturbed equilibrium or its equivalent disturbed body position which is occasioned also by these same stimuli acting through the medium of the subcortical centres. The actively disturbed body equilibrium, therefore, may be considered as representing the motor side, or motor expression, of the disturbed condition of both the cortical and subcortical centres.

Stimulation of the optic, auditory, and nerves of general sensibility give rise constantly to sensations of position when the body is at rest, and equilibrium can become actively associated with these sensations only when the body is under the tension of the act, either in the erect position or motion. Hence, in the passive condition of the body, sensations of position are developed through purely sensory stimuli, in the active state of the body, the same stimuli are in operation plus the motor adjustment necessitated by them, but without the development of sensations as far as this specific adjustment is concerned. In the latter instance of the body in motion, the equilibrium which is maintained may be termed dynamic, in the sense of being dependent largely upon motor effort; in the former instance, when the body is at rest, the equilibri-

um is almost purely psychical, and may be designated as static, being dependent upon normal sensory stimuli but without motor excitation.

The result of this excitation or adjustment is present in consciousness as the sensation of maintained equilibrium or body position, and if this adjustment is imperfect and continuous, then sensations of disturbed body position are the chief feature of consciousness. Hence, summing up the act of equilibration in its entirety, it would seem justifiable in considering it a psycho-physiological process, to define its psychical element as the consciousness of body position, while the muscular adjustment maintaining this position represents the motor element of the same process. Certainly, in vertigo we have, on the one side, the consciousness of disturbed body and spacial relationship, and on the other, the consciousness of unavailing motor efforts to rectify this relationship.

Most writers on the subject of equilibration and vertigo have emphasized the physiological side of the problem, and it has seemed to the author that the failure to recognize the importance of the psychical element has prevented the full understanding of the nature of vertigo. The fact that equilibration can, and ordinarily does occur, independent of consciousness, has led to the ignoring of the influence and connection of the cortex with the act. It seems to have escaped notice that the integrity of the act depends almost as much upon the condition of the cortical as upon the mesencephalic and cerebellar centres. Thus, if the psychical centre, or consciousness, is clear, equilibration is perfect; if consciousness is clouded or disturbed from the normal condition, then equilibration is, or may be imperfect, depending upon the character of the cortical disturbance. As far as equilibration is concerned, consciousness may become clouded in two ways: directly, by the reception of disturbed sensory stimuli yielding sensations of disturbed body position, as is the case of true

primary vertigo; or indirectly, by the occupation of the whole field of consciousness by other sensations to the exclusion or modification of its normal space and position attributes. This latter condition occurs under strong emotion, shock, pain, or the effect of an idea. The dizziness or loss of the sense of position which accompanies this condition of consciousness results, primarily, not from disturbed sensory stimuli, but from the more or less complete absorption of consciousness in some powerful impression made upon it. Consciousness is preoccupied, and the sensory stimuli fail to produce their natural effect; they are negated, and consciousness, therefore, is without its usual statical attributes. The vertigo associated with gastric disorders, cranial nerve crises, emotional states, or whenever present and not originally due to perverted position stimuli, can only be satisfactorily explained by recognizing the cortex as a factor in the function of equilibration.

The ground or basis for advocating the importance of the condition of the cortex in the act of equilibration, and as related to the production of vertigo, is the principle of psycho-physiology so ably developed among English medical writers, by Hughlings-Jackson, and supported by the anatomical and embryological researches of Flechsig, *viz.*, that all parts of the body have a representation in the cortical centres, and conversely, that these centres may exert an influence on all these parts. Also, in connection with this principle is the accepted fact that all ideas or sensations tend to express themselves, so that whatever the state of consciousness may be, there exists a constant tendency for it to be manifested outwardly.

The understanding of this principle of cortical influence, the writer believes, is the key to the explanation of all forms of vertigo which arise outside of the primary involvement of the special sensory stimuli which yield sensations of position.

A REPORT OF A CASE OF UNUSUAL EDEMA IN HEMIPLEGIA.¹

By H. A. HARE, M.D.,

Professor of Therapeutics in the Jefferson Medical College, Philadelphia, Pa.

The case that I desire to present this evening will be described in detail in a few minutes. I report it because the extraordinary edema of the hand, forearm and the lower and middle third of the arm is certainly an unusual complication in cases of hemiplegia. You will also notice from the history that this edema, which began in the hand, gradually spread up the arm, and that finally, when it ceased to spread, it was separated from the upper arm by a distinct line of demarcation; not that there was any discoloration of the skin, but that the swelling suddenly ceased as effectually as if a tight band were placed about the arm at that place.

The swelling, too, is far in excess of that arising from trophic changes in the arm, which is sometimes seen in hemiplegias. It is an edema quite as marked as that which is frequently seen in cases following phlebitis in convalescence from typhoid fever or parturition.

The picture which I show you illustrates the condition fairly well. An interesting point in her history is that, notwithstanding the fact that she is totally hemiplegic and that this symptom came on suddenly, there was no loss of consciousness with its onset.

The diagnosis as to the cause of the hemiplegia lies between hemorrhage, hysteria, embolism and thrombosis. That it is not thrombosis, I think, is proved by the manner of onset and by the fact that the woman's blood vessels are in good condition, except, perhaps, in the area of the brain in which the rupture may have taken place. Neither does

¹ Read before the Philadelphia Neurological Society, February 22, 1897.



it seem to me likely, from a study of her case, that the case is one of embolism, although an examination of her heart shows a mitral regurgitant murmur. It is, however, much more common to find embolism resulting from mitral stenosis than from mitral regurgitation.

With the report of the case there is a careful report in regard to her eyes, made by Dr. de Schweinitz, this report being practically negative. I am inclined, therefore, to believe that the case is one of hemorrhage, and I am quite confident that it is not hysteria.

A careful examination of her arm, with particular attention to its bloodvessels and nerves, fails to throw any light upon the case. There is no evidence of interference with either the venous or arterial circulation, nor are there any signs of a peripheral neuritis.

It is an interesting fact that the paralyzed leg is not in the slightest degree edematous. It may also be important to remark that this edema could not have resulted from pressure as the result of lying on the paralyzed part, as

she has been carefully nursed, and it is now a number of weeks since the edema developed. Because of the unusual character of the case, I asked Drs. Dercum and Spiller to see it with me, and they both agree that in their experience the case is unique.

Mrs. Georgianna Robertson; widow; aged, 46. Residence, Dearfield, N. J. American-born.

Admitted to Jefferson Hospital, Dec. 19th, 1897.

Family History.—Negative.

Personal History.—For three years has been under treatment for valvular disease of the heart.

Present Trouble.—For more than a month there has been failure of compensation as evidenced by swelling (edema) of the legs. On the night of Dec. 17th she was seized suddenly with paralysis of the right side of the body and aphasia. She was conscious, however, and has been up to date, Dec. 20th, 1896.

Jan. 13th, 1897. When Dr. Hare came on duty he noticed that there was no ptosis, no forehead paralysis—mouth drawn to left, typical facial palsy, limited to lower part of right face. Thyroid gland enlarged and somewhat pyriform. No other signs of goitre. Some wasting of right arm and leg of right side. Aphasia absolute. Skin reflexes normal on both sides. Hyperesthesia of left hand. Anesthesia of right hand. Analgesia to elbow on right forearm, decreasing to arm. No marked loss of temperature sense in right arm. Apex beat well marked and somewhat diffused; no thrill. Moderately well developed mitral systolic murmur. Faint aortic systolic.

Feb. 15th, 1897. Tongue is protruded all right; swelling of arm decreased. Great restlessness with moaning and groaning and holding left leg.

Feb. 16th, 1897. Aphasia not so marked. Rapid respirations, 48 per minute; pulse rapid and feeble.

Feb. 17th, 1897. Speech returned to fairly clear enunciation.

Feb. 20th, 1897. Drowsy and stupid. Tongue foul from lack of movement. Can protrude it very well. Urine 1010, acid, albumin 1/6 layer. No sugar. Hyaline and granular casts and renal epithelium. Eye—Movements of the eyes good in all directions (Feb. 17th, 1897). Right palpebral fissure wider than the left, 12 and 10 mm. respectively. Convergence good—left internal rectus is slightly weaker than right. As left eye diverges, right still maintains position of convergence. Pupils round and equal—pupils normal in reaction. Eyes very unsteady. Field examination in four meridians; intermediate meridians show similar contraction, but no quadrant loss. Central field apparently normal—certainly for red and green. Ophthalmoscopic.—R. E.—Vertical oval disk, grayish in deeper layers, no neuritis or atrophy—vessels anemic, about normal in size—slight perivasculitis—no hemorrhages. L. E.—Similar disk, grayer than on other side, and slight edema of fibre layer of etina.

Visual field measurements.—Outward, O. S. 40, O. D. 50; upward, O. S. 20, O. D. 30; inward, O. S. 40, O. D. 35; downward, O. S. 45, O. D. 50. Eye negative as to paralysis. Examination by Dr. de Schweinitz.

Jan. 18th, 1897. Patient began to recover speech, "yes," "no," "well."

Jan. 22nd, 1897. Recovery of speech gradually increasing.

Jan. 30th. Patient can speak several words, "I feel better," "No," "I want a drink," and such short phrases. Ecchymosis under right eye mostly gone.

Jan. 25th. Fingers of right hand (the paralyzed one) slightly edematous and swollen; some perception of pain in moving elbow; no recovery of motor power.

Jan. 27th, 1897. Dorsum of hand edematous; skin smooth and shiny; pits slightly; some tension; limited at wrist.

Jan. 30th. Swelling has gradually extended to the elbow of same character; limit sharply marked.

Jan. 31st. Elbow involved. Some sensory perception on handling arm.

Feb. 3rd. Four inches above elbow line of demarcation marked. Measurements, 4 inches above elbow, $9\frac{1}{2}$ inches circ.; 3 inch above elbow, $12\frac{1}{2}$ inches; 1 inch below elbow, $12\frac{1}{2}$ inches.

Feb. 17th. Swelling diminished; skin brawny; some slight and irregular desquamation; area of bluish color on outer anterior aspect of arm (right) just above bend of elbow and directed obliquely up and out. Hand still somewhat edematous.

Feb. 11th, 1897. Abdomen distended with gas; some pain; swelling of hand and arm better; not so edematous; thyroid much enlarged.

Feb. 22nd, 1897. Abdominal distention not so marked; thyroid a little smaller.

Note.---Before the patient's death edema was also noticed in the right lower extremity. An autopsy was obtained and the brain was found to be very edematous. In a horizontal section at the level of the superior part of the thalamus and striatum of the left side, there was a hemorrhage occupying the external capsule and lenticular nucleus. It appeared to be about the size of a hickory nut, and the brain substance around it was softened, more especially on the inner side of the focus. The anterior part of the posterior limb of the internal capsule was evidently involved in the softening, but the optic radiation, except, perhaps, its most superior part, appeared microscopically to be intact. In a section made horizontally through the left hemisphere, one inch below and parallel to the first section, there were no evidences of hemorrhage. All the basal arteries of the brain were atheromatous. The kidneys were greatly contracted.

CLINICAL CASES

Reported from the Clinic of Prof. M. Allen Starr, College of Physicians and Surgeons, New York.

FRIEDREICH'S ATAXIA.

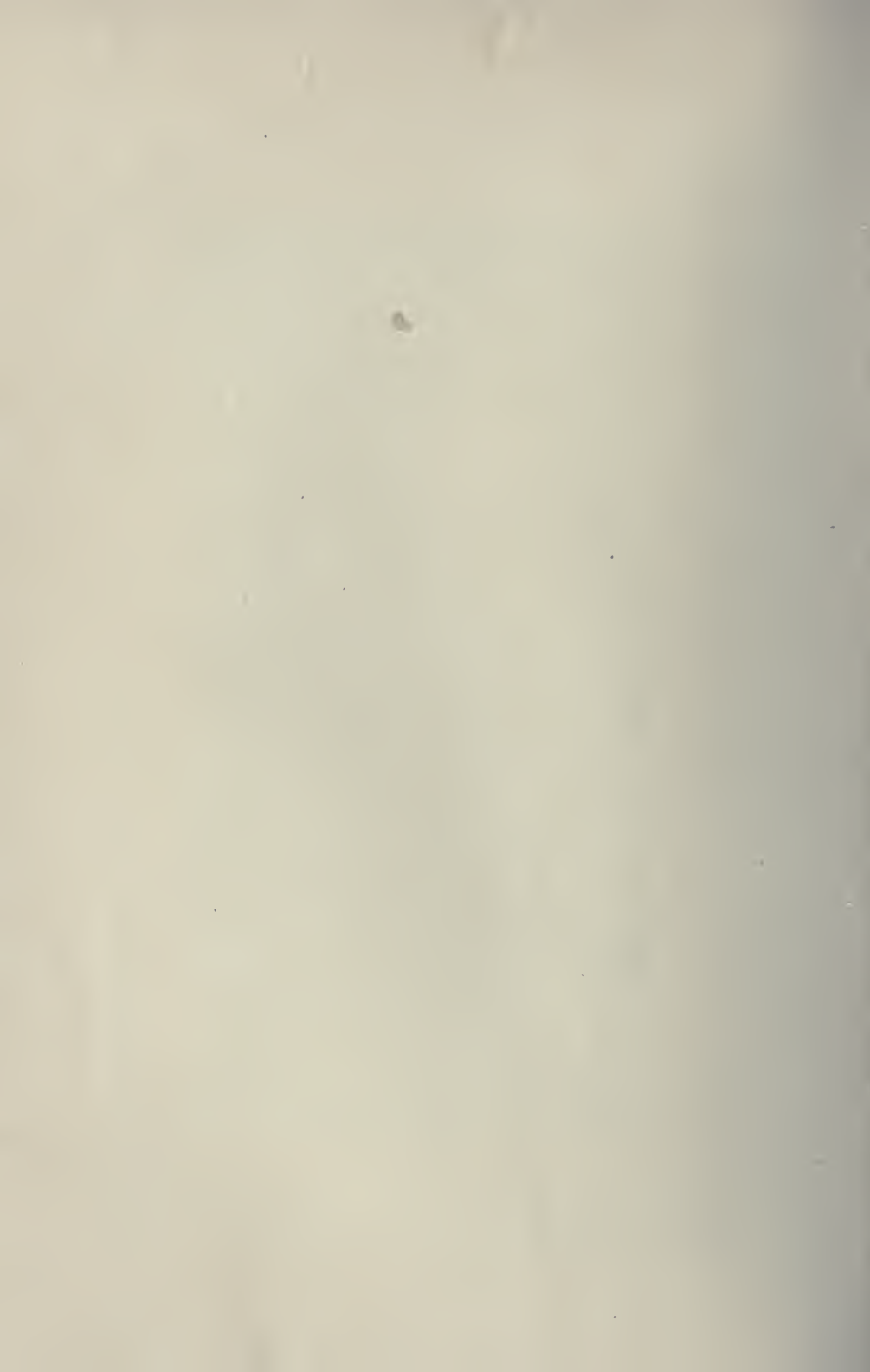
This disease is sufficiently rare to warrant the report of cases.

Case I. Susan J., aged thirteen in December, 1897, is the second of five children. Her father and mother are healthy, and no one of her grand parents, aunts or uncles are known to have had any form of paralysis. An older brother, however, suffered from this disease which developed at the age of eleven and rendered him a cripple until his death from diphtheria last year. The patient was a healthy child, though it was noticed that as a baby, while learning to walk, she had a somewhat waddling gait and was clumsy on her feet. Her present illness, however, did not begin until the age of eight, after an attack of measles. Since that time difficulty in walking has gradually and progressively increased, and one year after the beginning of the trouble in the legs it was noticed that the hands were used in a clumsy manner. During the past five years she has suffered some from pains which were supposed to be rheumatic, and has occasional retention of urine which has never, however, required the use of a catheter. She applied at the clinic on account of the unsteadiness of her gait in walking, and examination showed her gait to be markedly ataxic, the steps being irregular in length, the feet being placed too widely apart, but tending to overlap in walking unless corrected by voluntary movements to preserve her equilibrium, the feet dropping somewhat with the toes turning inward and, hence, being lifted too high from the floor in the act of walking. It



FIG. I.

Attitude of patient with Friedrich's disease. The feet are too far apart and the body is bent forward. The oscillation of the head prevented a clear photograph of it.



was difficult for her to stand still without swaying, and this swaying was increased by closure of the eyes. Her knee jerks were lost. There was considerable ataxia of the hands in voluntary movement, and in all the automatic acts of dressing, fixing the hair, etc., the ataxia was apparent. There appears to be no disturbance of sensation in any part of the body to touch, temperature or pain impressions. The irregularity in the action of the bladder is not constant but occasional. She frequently wets the bed at night. A slight scoliosis is present, dorsal with convexity to the left, but there is no evidence of Pott's disease. There is some unsteadiness of the muscles supporting the head, and in consequence peculiar nodding motions of the head are seen, both, held at rest and while walking. Her pupils react normally, both, to light and accommodation, and her optic discs are clear. Her mental condition appears to be good.

Case II. Paul K., thirteen years of age in January, 1898. He is the third of nine children, only three of whom are living, the others having died in infancy from diseases not of a nervous character. He has one sister older and one younger than himself, neither of whom is affected by the disease. He is, therefore, the only member of the family thus far affected. His father died of paresis one year ago. His mother has had several miscarriages. The probability of a syphilitic inheritance is therefore great. The boy has always been delicate, has had numerous children's diseases, has suffered for long periods from gastrointestinal derangement, and at the age of two had a large abscess of the neck. He has always been considered weak and feeble, and was never bright at school, it being remarked that he laughed at everything in a silly manner, and was not able to learn quite as well as other children; yet he appears fairly intelligent, is able to read and write, and, though having a dull facial expression, is not, apparently, weak-minded. His present illness began at the age of ten, very gradually, it being noticed that he was awk-

ward in his gait, would stumble in going up and down stairs, and at the same time became clumsy in the movements of his hands. About the same time his mother noticed peculiar nodding or oscillating movements of the head, a tendency to look downward a good deal of the time. She says that he has always wet the bed at night, but that this has become more frequent of late.

Examination showed a very marked ataxia of gait, it being impossible for him to walk a straight line. His body stoops somewhat forward, as shown in the photograph, and it is impossible for him to raise the toes or feet from the floor while standing on his heels. He lifts his feet too high in walking, his steps are of irregular length; there is a tendency of the knees to overlap, though this is corrected by voluntary efforts to preserve his equilibrium, the toes fall down and inward as the foot is raised from the ground, making it necessary to step high in walking, and any attempt to stand or walk with the eyes closed results in a fall. The feet are markedly misshapen, the instep being too high, the foot somewhat clubbed, and the great toe and all the toes to a less degree being overextended so that all the tendons stand out upon the back of the foot. This deformity is shown in the photograph, in which a normal foot of about the same size is shown for contrast. He is able to place the foot flat upon the floor which corrects to some extent the deformity, but does not affect the hyperextension of the toes. All voluntary movements of the feet and hands are extremely ataxic. There appears to be a tendency to hold the fingers in a flexed position, suggestive of a beginning claw-hand. There is no scoliosis. There is constant oscillation of the head of slight degree laterally and antero-posteriorly, and this oscillation is increased on efforts of walking. His knee jerks are very much exaggerated, but there is no clonus. There are no sensory disturbances to tests of touch, temperature or pain. Muscular sense is much impaired; there are no elbow



FIG. II.

The foot of the patient with Friedreich's disease contrasted with a normal foot. The high instep, the greater arch, and the retracted great toe are noticeable.



FIG. III.

Appearance of the legs and feet in Friedreich's disease. The slight ptosis is also visible.

or wrist jerks. His pupils react normally to light and accommodation. He has slight lateral nystagmus in both eyes on looking far to either side. Optic discs normal.

Case III. Female, aged eighteen in December, 1897. She is the only child in the family, and both parents are perfectly healthy, and there is no history of any similar affection to be found in any member of the family. Her disease developed about the age of six, after an attack of measles, and has been gradually progressing ever since, but its progress has been so slow that it is difficult to fix a date for the onset of special symptoms. It is known, however, that by the time she was thirteen, her walking was very difficult, and that she had by that time become awkward in her hands and unsteady in her head. Examination showed a rather thin, stupid-looking girl, with her head habitually held slightly forward and downward in a slight state of oscillation, which was increased by the act of walking. She seemed to have some difficulty in raising the eyes or in looking upward. This is evident in the photograph. There is no strabismus, but she has a slight lateral nystagmus on turning the eyes to the side, and the eyelids cannot be fully elevated, and the eyeballs cannot be rolled upward as far as normal. The pupils react to light and accommodation, and the optic discs are normal. She has marked ataxia of the hands on all movements, and very great ataxia of the legs in walking, so that the act is an irregular stepping one with characteristic deformity of the feet, tendency to drop foot with incurvation of the foot and bending forward of the entire body in the act of balance to a slight degree. She has a slight lateral curvature of the spine toward the right which has required the use of braces for about two years. She has no disturbance of the bladder or rectum and no sensory symptom of any kind. The knee jerks are lost. The awkwardness of her hands is very marked in the act of dressing. She appears to be dull mentally, takes very little

interest in things about her, and is rather slow in her speech, with some hesitation. Her mother, however, will not admit that she is weak-minded in any degree.

These cases are grouped together as they are fairly typical of Friedreich's ataxia. It is interesting to notice that two of them began after an attack of measles. Considering the frequency of this disease in children and the fact that it often runs through a family, affecting several members, the question may be raised whether too much attention has not been given to the supposed hereditary factor in this disease. I have records of three other cases of Friedreich's ataxia, and in none have I been able to ascertain any evidence of similar disease in a previous generation. As the disease does not affect the duration of life, and the existence of a chronic cripple in a family cannot be overlooked or forgotten, it seems to me evident that many cases do not rest upon an hereditary basis. Nor can it be stated with precision that this is a disease of maldevelopment, for in the majority of cases children have grown to the age of six or eight without any manifestation of the disease, and have learned to walk well. The degenerative changes, therefore, in the nervous system must supervene upon healthy nervous tissue, and while it may be regarded as probable that from some unknown reason the life period of some neurons may be unusually short in these cases, just as the life period of muscles is abnormally short in cases of dystrophy, yet it is manifestly wrong to speak of a congenital maldevelopment in these cases. That the degenerative process is not exclusively limited to the spinal cord is manifest from the existence of ocular and mental symptoms in all the cases. Nystagmus was present in all, a tendency to ptosis and manifest weakness of the superior rectus muscle was present in two. Marked mental dullness was present in all three. It is, therefore, probable that the influence which leads to the disease is one which affects the entire nervous system, and if, as seems very

probable, the occurrence of acute infectious diseases, notably measles, is the exciting cause, this disease may be in future classified with multiple sclerosis, as a nervous sequel of the infectious diseases of children.

The possibility of Marie's disease, in the second case, is admitted. As yet it seems impossible to draw a sharp line between Marie's disease and Friedreich's disease clinically.

77. SPLITTING THE KIDNEY CAPSULE FOR THE RELIEF OF NEPHRALGIA.

In the Medical News, Jan. 30th, 1897, Dr. George B. Johnson reports two cases in which violent pain, resembling attacks of renal colic, led to the operation of nephrotomy for supposed calculus, although the urinary symptoms were not characteristic. In neither case was stone found to be present. The kidneys, however, appeared to be too tightly enclosed in a tense capsule. In each case a free slit was made in the whole length of the kidney capsule. The pain was promptly abated by this procedure and never returned. The author concludes:

1. Nephralgia is not always associated with a demonstrable lesion.
2. When other evidences of kidney disease are wanting, the pain is perhaps due to a too-tight capsule.
3. Nephralgia may, and frequently does, simulate symptoms of gross tissue changes or presence of mechanical irritants.
4. When severe and persistent pain in the kidney exists, without other evidences of renal involvement, exploratory operation is indicated.
5. When inspection, palpation, and needle punctures fail to disclose a condition sufficient to account for the pain, the capsule should be freely opened.

SHIVELY.

Society Reports.

NEW YORK NEUROLOGICAL SOCIETY.

Stated Meeting, February 1st, 1898. B. Sachs, M.D., President.

Dr. Mary Putnam-Jacobi presented a boy of three years, who had begun to talk well when two years old. About September 2d, 1897, the mother noticed the left arm begin to tremble. A week later the child fell down in the street, and a wagon ran near, but not over him. A policeman insisted that the child had been injured, and he was taken to a hospital, where the doctors stated that he had a hemichorea due to fright. As the trembling of the arm had preceded the fall, it was hardly possible to attribute it to his fright. A little later, the child had had quite a severe attack of measles, with pneumonia, and during that time the tremor ceased. It was noted shortly afterward that the leg also trembled. Iron and arsenic were given freely, but with no benefit. On November 25th, there was then weakness in both the leg and arm, and these steadily increased. On January 3d, 1898, she had first seen the child. There was then a condition present which the mother had not noticed—i. e., a deviation of the right eye outward and a marked dilatation of the pupil and very slight reaction to light. Vision was good. At present there is a noticeable drooping of the left angle of the mouth. Another new symptom is an inclination of the head to the left side, with slight resistance on attempting to straighten it. The disposition of the child is good. His speech is indistinct, but this may be because he is so young. The spontaneous tremor in the leg has disappeared, but an attempt to walk sets up inco-ordinate movements in the leg. Sensation and electrical reactions are normal. The knee-jerk on the affected side is decidedly increased. The diagnosis seems to rest between hemichorea, with consecutive paralysis, post-hemiplegic chorea and multiple sclerosis. Two or three physicians had made

a diagnosis of tumor, but this seemed to be excluded by the absence of vomiting, convulsions, headache or alteration of character. The existence of ocular symptoms on the opposite side would seem to negative the diagnosis of chorea. A lesion in the inner part of the right thalamus, or under the aqueduct of Sylvius, would explain the symptoms. The extreme youth of the child would not exclude such a diagnosis, for several such cases were on record.

DISCUSSION.

Dr. L. Stieglitz said that two years ago he had exhibited to this society the brain of a child of two and a half years, with a tubercle in the right crus. In this case a very similar symptom complex had been observed, *i. e.*, left hemiplegia with a characteristic crossed third nerve paralysis. There was a very similar tremor for months, present both at rest and on movement, until total paralysis occurred. Gowers states, in his book, that solitary tubercle produces not rarely an intentional tremor. He had found recorded as many as 35 cases of disseminated sclerosis in young children. One should be careful not to base the diagnosis upon the presence of intention tremor alone. It would be interesting to know if there had been choked disk in the case just presented. He would venture to predict that the case would progress to a fatal termination, and that tubercle would be found. In his own case, the choked disk had developed only very shortly before death.

Dr. C. A. Herter said that he had seen the patient about a month ago. He had leaned toward the diagnosis just made by the last speaker. Two years ago he had had a case of crossed paralysis of the same general character. In view of the fact that tubercle of the brain is a much more common condition than multiple sclerosis in children, such a diagnosis was the more probable one. He was under the impression that the child's eyes had been examined with negative result.

Dr. Sachs said that he had seen this child about three months ago, when the condition had been quite different. On examination, he had found slight rigidity in the left extremities and increased reflexes in the upper extremity and in both lower extremities. The statement was made at the time that the examination of the fundus of the eye was entirely negative. At the time he had made a tentative diagnosis of post-hemiplegic tremor or a post-hemiplegic ataxic tremor. He thought now that it was exceedingly probable that there was a neoplasm in the brain. In a case published by him some time ago there had been very marked ataxic movements,

and the autopsy had shown a lesion in the crus. In all the cases of this kind that he had seen the ataxic tremor had been observed only on attempting to move the arm, whereas in this case the tremor is continuous.

Dr. Putnam-Jacobi said that the occurrence of measles and pneumonia after the beginning of the nervous symptoms, without the development of pulmonary tuberculosis, seemed to argue against the diagnosis of tubercle. If a tumor were present, as had been suggested, there should have been a total paralysis of the third nerve, whereas there was only an associated paralysis. Another point was, that there was no hemiplegia until some time after the development of the tremor.

LOCALIZED SYRINGOMYELIA.

Dr. L. Stieglitz presented a woman, thirty-eight years of age, who two years ago began to complain of pains in the left shoulder, arm and forearm. Shortly after this, she noticed some weakness in her left hand, and subsequently wasting of the muscles of the hand. The fingers then became contracted as at present. Examination in August, 1897, showed complete atrophy of the thenar, hypothenar, interossei and other intrinsic muscles of the left hand, and contractures of the long flexors of the fingers, especially of the three ulnar fingers. Along a narrow strip of the inner surface of the left arm sensation was disturbed. The left eye is markedly sunken, the left pupil is small and remains so in a dark room. It does not dilate under cocaine, but is dilated by atropin without difficulty. He considered this a case of very marked localized spinal lesion in the anterior part of the left side of the spinal cord. He thought an inflammatory condition, such as myelitis, could be excluded. There was also no history or evidence of syphilis, and she showed no change under specific treatment. This clinical picture might be produced by a neoplasm at the level of the anterior horn, or by gliosis. She had been under observation for six months, and had developed practically no new symptoms. He was inclined to regard the condition as one of syringomyelia, localized for the present at the level of the first dorsal root.

In connection with this case he exhibited a classical example of syringomyelia, in which there was the same condition of the eye. This patient, twenty-one years of age, is a porter by occupation. Two or three years ago

he had first noticed a stiffness of the left hand, and this had become steadily worse, and the hand always felt cold. Examination showed the left shoulder to be considerably higher than the right, and a marked scoliosis to the left in the middle and upper dorsal region; almost complete atrophy of the small muscles of the left hand, and marked atrophy of the muscles of the left forearm, arm and shoulder. When the skin of the left hand is injured, the wound heals very slowly. There is an analgesia of the entire left arm, and of a large part of the left chest and scapular region. The temperature sense is less affected than on the right side. The left palpebral fissure, the left eyeball, and the left pupil are smaller than on the opposite side. The left pupil does not dilate under cocain, but does so readily under homatropin. The speaker said that this condition of the eye was not uncommon, but was sometimes overlooked when both eyes are affected. A very excellent point in the differential diagnosis was the test with cocain.

DISCUSSION.

Dr. C. L. Dana said that some years ago he had had a patient with a very typical kind of progressive muscular atrophy. It ran the usual course, and terminated fatally. In the early stage there were precisely the same conditions of the eye, so that he had always considered this part of the usual symptomatology of the disease. For that reason, it seemed to him that the diagnosis of progressive muscular atrophy was admissible in the case just presented. The eye symptoms could hardly be of any particular value, except in localizing.

Dr. Stieglitz said that he could not positively exclude progressive muscular atrophy. Before the clinical picture of syringomyelia had been well known quite a number of cases were diagnosed as progressive muscular atrophy; moreover, syringomyelia often begins without sensory symptoms, because the gliosis commences in the anterior part of the cord.

UNILATERAL REFLEX IRIDO-PLEGIA.

Dr. W. M. Leszynsky said that the term "unilateral irido-plegia" was applied to an ocular condition in which one pupil does not react directly to light, while its reaction in convergence is preserved. The other pupil reacts normally. The pupil may be either dilated or contracted, or both pupils may be dilated. It is usually unaccompanied

by any interference with vision, or changes in the fundus. It might also be called a unilateral Argyll-Robertson pupil. Absolute iridoplegia of recent origin had been known in a few instances to disappear under the administration of mercury and iodide of potassium. It had been erroneously inferred that this unilateral form of iridoplegia is associated with tabes. The patient presented, a woman of thirty-eight years, was first seen by him in December, 1896. Her second husband had had syphilis, some years before marriage, and had since then given abundant evidence of the disease. She had never been pregnant after this marriage. The patient herself had been somewhat intemperate. Her left pupil is larger than the right, and she says that this has been so for at least three years. All of the external eye muscles act normally. There is none of the usual evidence of syphilitic infection, but the other signs and symptoms seem to warrant the diagnosis of cerebrospinal syphilis. She was improved somewhat by anti-syphilitic treatment. At the second examination it was found that both patellar reflexes were lost. In January, 1896, the pupils were found to be the same. This case could be considered as a typical one of tabes, or one of cerebrospinal syphilis.

He had found that only seventeen other cases had been reported up to date. From a study of these it would be seen that in nine there was a definite history of previous syphilitic infection, and in four the nervous manifestations justified the suspicion of antecedent syphilis. In thirteen cases the left pupil was affected; in eleven the iridoplegic pupil was dilated. The condition of vision or refraction seemed to have no bearing on the condition of the pupil. Apparently, unilateral iridoplegia is a very rare condition, though more systematic examination would probably show that it is more frequent than is now supposed. In three unrecorded cases of tabes he had seen unilateral iridoplegia at the first examination, but this had disappeared in a few weeks. He was inclined to think that in his patient when the pupil became affected there was a sudden ophthalmoplegia interna, and that the fibres had only partially recovered, thus leaving the pupil in its recent permanent condition.

The reader believed that the ciliary and sphincter nuclei are separate, and have independent muscular fibres. His conclusions were: (1) That unilateral reflex iridoplegia

is a condition which may arise in tabes or parietic dementia, being confined to one side for an indefinite time before the other pupil becomes affected; (2) that it often occurs in cerebrospinal syphilis, and as a remote result of disease or injury of the third nerve; (3) that it is always indicative of degeneration of the oculo-motor nerve; and (4) that the lesion is situated in the centrifugal portion of the reflex mechanism, as shown by its occurrence with, or as a consequence of, oculo-motor paralysis.

Dr. Carl Koller said that unilateral loss of light reflex is not such a rare condition as might perhaps appear from neurological literature. Of course, the loss of reflex to light is brought to our attention in two ways—one during neurological examination, and the other when the patient comes to the ophthalmologist complaining of symptoms. The latter are more frequent. He called to mind three cases, observed in private practice. Two of them had been followed for eight years. These patients had come with the pupil dilated and complaining of loss of accommodation. In the beginning, with the loss of reflex there had been also a loss of the power of accommodation. This had returned in part after a time. In two cases the loss of reflex appeared in the other eye, and also without loss of accommodation. From his own experience he would be inclined to believe that it was a nuclear affection, (1) because the accommodation is but slightly interfered with, and (2) because in most of the cases the nucleus on the other side becomes subsequently affected. In the majority of cases he believed syphilis to be the cause.

Dr. Herter thought the conclusions reached by the reader of the paper were justified by the facts. In one or two instances he had noted this one-sided irido-plegia, and had been puzzled by it. He had looked upon the lesion as one of syphilitic origin.

Dr. Sachs thought a difference in the action of the pupils not uncommon, and that for this reason it had not been oftener recorded. It seemed to him that unilateral reflex irido-plegia was invariably specific, and, indeed, a very important diagnostic symptom. Its presence had caused him often to suspect specific disease. Unilateral immobility, and especially double complete immobility, are characteristic signs of syphilis. He did not see how it could be anything else than nuclear in its nature.

Dr. Leszynsky, in closing, said that the experience of Dr. Sachs was opposed to that of a number of observers, who had studied a large number of cases of pupillary phenomena without finding more than a few cases.

PHILADELPHIA NEUROLOGICAL SOCIETY.

December 20th, 1897.

President, Dr. Charles W. Burr, in the chair.

Dr. James Hendrie Lloyd presented a case of

ASTASIA-ABASIA.

The patient, a female, had much difficulty in standing and walking. Under suggestive therapeutics her condition had improved.

DISCUSSION.

Dr. Wharton Sinkler thought that the gait and whole attitude of this patient were very characteristic of the hysterical affections of gait. He stated that these vary greatly in degree and character, but this case he regarded as a type of the so-called astasia-abasia. In hysterical disturbances of gait there is usually a tendency to pitch to one side or backward, but the patient seldom falls or completely loses her balance, unless there is some one near to catch her.

Dr. David Edsall, by invitation, read a paper on

DISSOCIATION OF SENSATION, OF THE SYRINGOMY- ELIC TYPE, IN POTT'S DISEASE.

DISCUSSION.

Dr. Spiller stated that a few years ago it was thought that syringomyelia could be positively diagnosticated by dissociation of sensation; but, as Dr. Edsall's case and many others show, the difficulty in making this diagnosis increases. This dissociation of sensation occurs in tumors of the cord, in tabes, in disseminated sclerosis, in hysteria, in neuritis, and has been observed in the peculiar disease of the spinal column reported by v. Bechterew and Strümpell.

He did not think that the diagnosis of a tumor in this case could be made with any certainty. A spinal growth might be present and cause few symptoms, inasmuch as a tumor of the oblongata may be latent for some time. It seemed to him that all the symptoms could be produced by a lesion of the posterior roots, or by compression of the cord. This sug-

gested the interesting theory of Goldscheider in regard to sensation, according to which pain is simply a summation of tactile impulses. Where pain sense is lost, and tactile sense is present, as in tabes, the posterior roots, though degenerated, permit the transmission of certain impulses, but not of a sufficient number to cause the perception of pain. This theory has, by no means, been accepted by all neurologists.

Dr. Hinsdale remarked that, about three years ago, Dr. Lloyd had shown, at one of the meetings of the Society, two cases of traumatic lesions of the spinal cord with syringomyelic symptoms. He thought that it is quite possible that in such cases there is extravasation of blood—a hæmatomyelia, which, after a time, is absorbed, but gives temporarily the dissociation symptom. This symptom does not persist, but causes a good deal of difficulty in forming a diagnosis while it lasts.

Dr. Eshner said that the fact that the sensory symptoms receded appeared to be against the probability of the existence of a tumor within the cord. One would not expect even a tuberculoma to diminish in size or to disappear within such a short time.

Dr. Burr spoke of the frequency of new growths, as shown by the necropsy, which have not been diagnosticated during life.

Dr. Edsall thought that it was quite possible that a tumor might have formed since the caries developed. In some tuberculous tumors that have been reported the growth has been very rapid. The peculiar distribution of the hypalgesia and thermal anæsthesia, and their extension to lower parts of the body, had led him to suspect the presence of a new growth. These could be better explained by the presence of a tumor than by any other means, provided the tumor originated in the centre of the cord and grew outwards.

Dr. W. E. Hughes, by invitation, reported

TWO CASES OF TUMOR OF THE BASE OF THE BRAIN,

and exhibited one of the specimens. A growth, the size of a hen's egg, was situated in the pituitary body. No symptoms of akromegaly had been observed during life.

DISCUSSION.

Dr. Hare desired to know how much of the pituitary body was left intact. If it is true that akromegaly often arises from disease of the pituitary body, it may be that when a certain

portion of this body is left intact the symptoms do not develop. In the same way, if the pancreas is destroyed diabetes may appear, while if a portion is left pancreatic diabetes may not develop.

Dr. Hughes was unable to say how much of the pituitary body was involved.

Dr. A. A. Eshner made

A FURTHER CONTRIBUTION UPON A CASE OF MEN-
INGITIS.

The report of this case was published in the *Journal of Nervous and Mental Disease* for March, 1897, p. 167. The patient died in convulsions, and a necropsy was made by Dr. Cattell.

On removing the calvarium the dura mater was not found adherent to the bone in this situation. The vessels generally were greatly injected, and the capillaries were somewhat more prominent than normal. All of the sulci in the Rolandic area on both sides exhibited a grayish-white, delicate, fibrous reticulum, which was slightly elevated on account of the presence of subjacent fluid. There was no indication of any process suggestive of recent bacteriologic involvement. The appearances were rather those of chronic fibrous thickening of the pia-arachnoid, with moderate adhesion to the brain surface. The blood vessels forming the circle of Willis were thickened, and gaped when cut. Many of them contained small fibrous nodes. The fibrous thickening of the meninges previously referred to was especially pronounced in the neighborhood of the basilar artery and upon the pons and medulla, the nerves coming off from which passed through the thickened membrane.

The under surface of the cerebrum, the olfactory, and the temporal regions were remarkably free from thickened membrane, which was, however, prominent in the fissure of Sylvius along the course of the middle cerebral artery. About three-quarters of an inch from the point of origin of the left middle cerebral artery was a small, well-formed sacculated aneurism, about the size of a small pea. The island of Reil and the retroinsular convolutions displayed no abnormality, other than atheroma of the blood vessels, and a small clot of blood on the right

side, apparently contained within a miliary aneurism. There was considerable œdema in the neighborhood of the optic chiasm, and the optic nerve appeared flattened from above downward and diminished in size, from thickening of the overlying adventitious membrane.

The left lobe of the cerebellum was the seat of an enormous hemorrhage, which had ploughed its way into the fourth ventricle, and thence through the iter into the third and also into the left lateral ventricle. So enormous was this extravasation that a considerable clot of blood was found in the posterior horn of the left ventricle. The foramen of Munro and the adjacent nervous tissues were torn apart and replaced by a bloodclot. Sections through all parts of the brain failed to disclose any other seat of hemorrhage, recent or remote. The injection of some portions of the arbor vitæ of the cerebellum was especially pronounced.

Concerning the other organs, it seems only worth saying that the kidneys exhibited an extremely slight degree of parenchymatous inflammation, while the heart was somewhat enlarged, with healthy valves and orifices, and the lungs were œdematous and emphysematous.

Owing to the conditions under which the autopsy was held, it was not possible to obtain even the smallest portion of tissue for more careful study. It could not be decided from the macroscopic appearances whether or not the upper portion of the spinal cord exhibited any changes, particularly in its posterior columns.

Dr. E. Schreiner presented

A CASE OF ARSENICAL NEURITIS FOLLOWING THE
TREATMENT OF CHOREA WITH FOWLER'S SOLUTION.

The patient, a female of ten years of age, was seen April 17th, 1896, in her second attack of chorea. She was placed on Fowler's solution, beginning with five drops three times daily. This was increased to twelve drops, when symptoms of the physiological action were observed, and the quantity was reduced. The total amount taken was two or three ounces. She remained under occasional observation until June 5th, when she showed loss of power in the arms

and in the legs, more marked in the latter, with areas of pigmentation over the joints, some atrophy of the leg muscles, loss of patellar and plantar reflexes, diminution of tactile sensation, and marked reaction of degeneration, viz., complete loss of irritability to the faradic, and very slight response to strong galvanic currents.

There was a history of pain in the calves of the legs and of swelling below the knees two weeks before the patient's admission to the hospital. The only sensory symptom present on admission was some soreness in the course of the radial nerves.

Motor power and sensation improved, the latter more rapidly than the former, so that the patient finally was able to walk, though she presented the steppage gait. At present, December, 11th, 1897, the electrical reactions in the muscles of the legs and forearms are absent, and the muscular power, especially in the extensors of the leg, is imperfect, causing foot drop and steppage gait, but sensation is normal.

DISCUSSION.

Dr. Charles K. Mills said that this case interested him, especially in connection with the second question referred to, that is, as to the nature of the pathological changes which are probably present, and as to the names which should be used in speaking of cases of this kind. He thought that the time had come for us to revise our nomenclature of some of these toxic and infectious diseases of the nervous system. We certainly have very different types of cases which, without much thought, we class under the same names. Formerly, more than at present, these cases were spoken of as instances of myelitis; now we are more inclined to speak of them as cases of peripheral neuritis. It is to this point that he wished to ask attention.

He referred to a man who several years ago presented signs of arsenical poisoning. He got well after a time and left the hospital. Recently he came back with conditions similar to those referred to to-night. He had been addicted to the use of alcohol, and had been working as a painter, largely in white lead, for six or seven years. He had double foot drop, lost knee jerks, some irregularly disseminated disturbances of sensation, particularly dissociated anæsthesia, slight paresis of the upper extremities, and a little circumscribed pain in the limbs. Dr. Mills stated that a short time ago this case would have been looked upon by some as one of multiple neuritis; earlier, as one of myelitis; or, perhaps, it might have been

regarded as a case which showed a concurrence of neuritis and myelitis.

The point which he wished to emphasize is that we need a new designation for these cases. We need to reform our nomenclature and classification of cases in which, as the result of an infection, or a toxæmia, or the direct action of a metallic poison, we have symptoms such as were reported to-night. The only sensory evidences of true neuritis in this case were the pain and hyperæsthesia, which, however, were not marked and were circumscribed; although the paralysis and loss of electrical reaction might also be regarded as due to neuritis.

In naming these diseases the idea should be enforced that the toxine, infection, or poisonous substance has set up a process which has led to the destruction of the peripheral neuron. Something circulating in the blood of the patient attacks the nerve cells—both cell body and processes, poisoning and destroying them, but not necessarily giving rise to inflammation. It is difficult to have clear ideas with regard to myelitis and neuritis on account of the different uses of terms.

Dr. Mills said that if he were asked what he would call cases of this kind, it would be a little difficult to say. We know that the chief evidences of inflammation are found in such conditions as hyperæmia, swelling and round cell infiltration. If these were present, we could properly class the disease as inflammatory. In their absence it is doubtful whether they should, or should not, be regarded as inflammatory affections. In reported cases both the peripheral nerves and the ganglion cells in the anterior horns of the cord have been found destroyed, or degenerated, without the usually accepted evidences of inflammation. It is, therefore, an open question whether these cases should be designated as neuritis, myelitis, or myeloneuritis; or whether, indeed, it would not be better to invent an entirely new term for them. Provisionally, he suggested that they should be called cases of *peripheroneuronal degeneration*, with the prefix of *acute*, or *chronic*, according to the intensity and rapidity of the destructive process.

Dr. Wharton Sinkler thought that Dr. Mills was going backward if he advocated the theory of myelitis in cases of toxic poisoning. It certainly is impossible to explain many of the curious symptoms present by regarding the lesion as located in the cord. It seemed to him that in a case like the one reported by Dr. Schreiner the view of peripheral neuritis is sufficient to account for the symptoms. In myelitis there is not apt to be as great loss of electrical irritability, nor is there the peripheral pain, hyperæsthesia and foot drop.

Dr. A. E. Taylor alluded to the recent address of Virchow upon inflammation, and, commenting upon our ignorance of

many aspects of that process, expressed doubt as to whether the absence of round-celled infiltration could be relied upon as a criterion of the non-inflammatory nature of a pathological condition. The relations of inflammation to the interstitial and parenchymatous tissues would be clear if we knew exactly what inflammation always is, and what the parenchyma and interstitial tissues are.

Dr. Spiller stated that in many cases we are at present utterly unable to say whether a poison exerts its influence, primarily, on the nerve fibre or the cell from which this arises. He referred to the selective power of certain poisons for certain nerves, as seen in saturnine, alcoholic, diphtheritic and other forms of poisoning. He stated that probably all reported cases of recovery from tabes, such as are seen after diphtheria, are examples of pseudo-tabes, due to inflammation of sensory nerve fibres, and that certain poisons seem thus to pick out one set of fibres, even within a nerve, to the exclusion of others. He referred to the many experiments which have shown that, when a motor nerve is cut, the cell with which it is connected degenerates very rapidly. This degeneration may occur within a few hours after section of the motor nerve, and there is no reason for believing that these cellular changes are of an inflammatory character. They are changes in the chromophilic elements. He said that he had recently studied a case of compression of the cord, high up in the cervico-thoracic region, in which the direct cerebellar tracts were degenerated below the lesion, and the cells of the columns of Clarke had entirely disappeared. This, however, was in the nature of a retrograde degeneration in sensory fibres. He spoke of a case of acute neuritis, which he was then studying, and stated that the changes in the nerves were very great, and that the cells within the spinal cord presented chromatolysis.

Dr. Hare said that the fact that different poisons attack different parts of the nervous system is one which hardly admits of debate, and also that we have a pretty well-grounded idea that these poisons produce a primary neuritis, or some similar change. Many of the changes which occur afterwards, and appear to be centred in the higher nervous system, are, perhaps, also due to the poison, or, perhaps, due to what may be called an ascending degeneration, a degeneration secondary to the peripheral degeneration.

He wished to know from Dr. Schreiner whether he noticed that the pigmentation in his case had any relation to the distribution of the anæsthesia, and whether these areas of pigmentation were peculiar in localization. It was taught at one time that the areas of pigmentation and anæsthesia are in relation to the joints. In arsenical neuritis, for example, it has been said that the anæsthesia in the upper extremity has a

tendency to stop at the distal joint; if it goes beyond this, it stops at the elbow.

Dr. Mills said that he did not believe that the points which he had made had been met. He, of course, admitted that in many cases there is widespread peripheral involvement. In a case such as Dr. Spiller mentioned, where we find the peripheral nerves degenerated, and the cells in the anterior horns just as seriously degenerated, to his mind nothing justified us in calling these cases either neuritis or myelitis. If the law is true that pathological processes tend to follow in the line of physiological action, it is more probable that the toxic influence would be exerted from the centre toward the periphery in a motor nerve than the reverse. In any case, it must be acknowledged that the entire neuron is usually implicated. He desired to know from Dr. Spiller the intensity of the changes in the case he had referred to.

Dr. Spiller replied that he had examined the peripheral nerves, but was unable to say from "teased" preparations whether the fibres were inflamed or degenerated. He thought that it is very difficult to distinguish between the two conditions as regards the changes found in the nerves. He had noticed that the axis cylinders were destroyed, that some of the sheaths were swollen and filled with an increased amount of protoplasm and degenerated myelin, or else contracted and empty, and that the medullary substance was much enlarged. His examination had not yet revealed engorgement of vessels and round-cell infiltration. He said that the chromophilic elements in the nerve cells from which these fibres arose were broken up, and that the nucleus was displaced. He was unable to decide whether the cell was primarily or secondarily affected, inasmuch as similar lesions have been noted in both primary and secondary degeneration.

Dr. Schreiner stated that his reason for describing this as a case of neuritis was the fact that in recent literature cases of arsenical paralysis are described as neuritis, and because in three cases, the pathology of which he was able to look up, degenerative changes were found in the spinal nerves.

He stated in reply to the question of Dr. Hare that the pigmentation in the axilla was confined by the anterior and posterior borders of the axilla. At the elbow it formed a rather irregularly oval-shaped patch, extending above and below the crease of the elbow joint. There were no areas of pigmentation at the wrist joint. The pigmentation was limited by the boundaries of the popliteal space, and extended irregularly over the ankle.

Periscope.

With the Assistance of the Following Collaborators:

CHAS. LEWIS ALLEN, M.D., Wash., D.C. R. K. MACALESTER, M.D., N.Y.
J. S. CHRISTISON, M.D., Chicago, Ill. J. K. MITCHELL, M.D., Phila., Pa.
A. FREEMAN, M.D., New York. H. PATRICK, M.D., Chicago, Ill.
S. E. JELLIFFE, M.D., New York. JOSEPH SAILER, M.D., Phila., Pa.
WM. C. KRAUSS, M.D., Buffalo, N.Y. HENRY L. SHIVELY, M.D., N. Y.
W. M. LESZYNSKY, M.D., New York. A. STERNE, M.D., Indianapolis.

ANATOMY.

55. ON THE CHROME SILVER IMPREGNATION OF FORMALIN-HARDENED BRAIN. J. S. Bolton (Lancet, i., 1898, p. 128).

The author has obtained excellent Golgi preparations from specimens fixed in formalin, 5 per cent., for a period of from two to twelve months. His results were less satisfactory if the formalin hardening was less than two weeks. Specimens should be cut preferably one-eighth of an inch thick. After fixation the specimens are transferred to a bath of 1 per cent. ammonium bichromate, where they may remain for from a few hours to five days, after which time the impregnation deteriorates the pictures obtained. Other chromic acid preparations gave less promising results. After the proper time of immersion in the bichromate the pieces are transferred, after rinsing in distilled water, into 1 per cent. silver nitrate, where they remain from sixteen to twenty-four hours. A longer immersion in the silver did not hurt the impregnation. The specimens are then hardened in 60 per cent. alcohol for a few hours, dried and imbedded in melted paraffine without soaking and cut, cleared and mounted in balsam without cover slip. JELLIFFE.

56. BEITRAEGE ZUR STRUKTUR DER NERVENZELLEN UND IHRE FORTSAETZE (Contributions to the Structure of the Nerve Cell). Hans Held (Arch. f. Anat. u. Physiologie, Anatomische Abtheilung, No. 2, 1897, pp. 3, 4).

This is one of the most important contributions to the finer histology of the nerve cell that has thus far appeared.

The author first takes up the question of the significance of what have been termed the "chromophylic" granules. Held has already claimed that these granules, described more particularly by Nissl, were arte-facts due to the fixative solutions used, and in his previous contribution in the same journal, 1895, gave a number of experimental proofs to support his assertion. The present communication is an answer to Lenhossek's criticism of his earlier paper, wherein Lenhossek states that the granules have been observed by him in the fresh specimens. The author adopts the idea that the Nissl granules are brought out by means of acid fixatives, and shows that in spinal cord fixed with weak alkaline solutions (5 to 40 per cent. NaOH was used) the large cells in the anterior horns were completely lacking in these characteristic bodies, and that when he fixed with alcohol (which

is nearly always acid) or alcohol plus a small amount of acid, acetic usually being used, the granules were very conspicuous. The author further considers the standpoint of physiological chemistry, which teaches that the reaction of living nervous tissue is alkaline, but that almost immediately after death the reaction becomes acid, sometimes within a few minutes. This development of acid is, in the author's mind, the cause of the formation of the Nissl chromophylic bodies.

The paper further considers the erudite questions of the structure of protoplasm, the nerve cells in particular being the ground debated upon. Held is inclined to adopt the reticulum theory, somewhat modified from the original Fromann point of view, and rejects the filar theory of Fleming. The article is one that cannot be omitted from the range of the neurologist's reading if he is interested in the problems of structure and the interpretation of microscopical pictures.

JELLIFFE.

57. URBER VARIATIONEN IM VERLAUFE DER PYRAMIDENBAHN (On Variations in the Course of the Pyramidal Tract). A. Hoche (Neurolog. Centralblatt, 16, 1897, No. 21).

The author contributes to this subject the anatomical findings in a case of glio-sarcoma, situated in the central convolutions, causing descending degeneration, in the pyramidal tract; the course of the latter was found to vary from that usually observed. Examination by Marchi's method showed that in the peduncle, pons and medulla the region of the left pyramid was exclusively degenerated.

The decussation occurred at the normal level, but a portion of the fibres passed down into the opposite anterior tract.

This decussated anterior tract extended throughout the entire cervical region of the cord, and disappeared at the level of the first dorsal root.

Another unusual occurrence was the extension forward of the area of degeneration beyond the ordinary region of the lateral pyramidal tract, which normally does not reach beyond a line drawn through the lateral horns.

At the II. cervical in Gower's tract a bundle was observed, which showed itself throughout the cervical cord as an irregular figure.

Throughout the dorsal cord the crossed pyramidal tract extended also too far forward, and even in the lumbar region the pyramidal area was greater than usual.

The direct pyramidal tract showed a hook-like formation as low down as the dorsal region.

MEIROWITZ.

58. ON THE ENDOGENOUS OR INTRINSIC FIBRES IN THE LUMBO-SACRAL REGION OF THE CORD. A. Bruce (Brain, 20, 1897, p. 261).

The author discusses the fibres found in the posterior columns of the cord in the lumbo-sacral region, termed by Marie the endogenous fibres. These fibres form two very well-marked tracts, one lying in the anterior part of the posterior column, in close opposition to the posterior cornu, commissure and septum: the second in immediate relationship to the posterior median septum, and in part to the posterior surface of the cord. The first tract, the *cornu-commissural tract* (Hinterstrangsfeld); the second, termed by the author the *septo-marginal tract*, "Edinger's medianisches Hinterstrangsfeld."

These fibres do not degenerate upward according to the author, being spared in locomotor ataxia; but they do suffer degeneration in conditions which induce degeneration in the cells of the posterior horns.

1.) The *cornu-commissural tract* extends through the whole of the lumbo-sacral region, being traceable from the lowest dorsal segments

to the extreme tip of the conus medullaris. At the level of the lower lumbar region it attains its greatest size, and diminishes above and below this level. It lies throughout in close relation to the posterior commissure and, in the main, to the anterior part of the inner margin of the posterior horns. Posteriorly the tract has no definite margin. Its outer portion merges gradually into the part of the column behind it, Flechsig's middle root zone. The fibres which compose it can be seen entering it from the gray matter of the posterior cornu, and almost exclusively from that of the same side.

2.) The *septo-marginal tract* forms a racquet-shaped area or triangle, two of its sides being applied to the posterior median septum and to the periphery, respectively, and the third forming a somewhat indefinite margin between the degenerated and undegenerated parts of the cord, the anterior angle seemingly extending anteriorly to the cornu-commissural tract.

With reference to its origin, the evidence is still insufficient; the tract originates in part in the higher segments of the cord, gains greatly in bulk about the level of the first sacral segment, and terminates in the lower sacral and the coccygeal region, by sending fibres along the posterior median septum into the gray matter near the central canal, where they are lost among the other fibres in this part.

The author believes that in some parts of its path this is the same as Flechsig's "oval field." The tract is mainly descending, but Spiller and Dejerine have shown it to contain some ascending fibres.

Their function is undecided, but the evidence seems to point to their being connected with the lower organic reflexes. The paper is well illustrated and contains a partial bibliography. VOGEL.

59. ZUR LEHRE VON DEN SECUNDÄREN DEGENERATIONEN IM RÜCKENMARKE. (Secondary Degenerations in the Spinal Cord). By Dr. B. Worotynski (Neurolog. Centralblatt, 16, 1897, No. 23).

Experiments were made upon dogs, in which total and hemi-lateral sections of the cord at different levels were practiced. In all, 18 experiments were performed, the animals surviving from 1 to 127 days. The author's results are summarized as follows:

1. Secondary degeneration of the individual systems in the spinal cord of dogs does not develop synchronously; the fibres of the posterior columns of Löwenthal's bundle degenerate first; then those of the direct cerebellar and of the antero-lateral tracts; finally, the fibres of the pyramidal tracts. In the human brain the same order seems to be followed.

2. Degenerative processes, once begun, develop rapidly, almost simultaneously throughout the entire extent of the tract.

3. In the columns of Goll and Löwenthal the degenerative process attains its maximum severity, which may be observed by the methods of Marchi, in the course of the second week following section of the cord; in the direct cerebellar and antero-lateral bundles, in the course of the third week; in the lateral pyramidal tracts it is still possible at the end of the fourth week to observe a general intensification of the degenerative process.

4. With Weigert's method the secondary degenerations, even three weeks after section of dog's cord, are hardly to be seen.

5. The order in which the secondary degenerations of the different columns show themselves corresponds approximately to that in which the systems receive their sheaths during development.

6. Kahler's law on the arrangement of the root fibres in the posterior columns can be accepted absolutely. The same law holds good with reference to the human brain.

7. Throughout the entire cord up to the cervical region the columns of Goll receive their fibres from the posterior roots. In the cervical cord the posterior root fibres enter the columns of Burdach.

8. The descending system of the posterior columns consists principally of myelogenic fibres. The bundles of descending fibres in the posterior columns, described by different authors under various names, belong to one and the same system, which at different levels of the cord changes its position and form.

9. In hemi-sections of the cord degenerations of the columns of Goll, Burdach, Flechsig, Gowers and Löwenthal are observed on both sides. The decussation of the columns of Goll and Burdach, and partly of Flechsig, takes place on the posterior commissure. The fibres of Gowers' and Löwenthal's bundles cross over principally in the anterior commissure.

10. The antero-lateral (Gowers') columns in dogs extend forward as far as the anterior fissure of the cord, and partly enter the anterior columns.

11. Gowers' and Flechsig's columns must be considered as belonging, anatomically, to one and the same system. A portion of both tracts terminates unquestionably in the cerebellum in the region of the nucleus dentatus and nucleus tecti. Decussation of part of these fibres occurs, in all likelihood, in the upper cerebellar vermisiform process.

12. Descending degeneration of Gowers' and Flechsig's columns appears questionable. Such described degenerations must be attributed to the fibres of Löwenthal's bundle.

13. The existence in man of a separate descending system in the antero-lateral tract may be considered as proved.

14. The descending degeneration of the intra-spinal anterior roots from the point of section may be best explained by the passage into the former of fibres from Löwenthal's bundle.

15. The ascending degeneration of the intra-spinal anterior roots is occasioned by the passage into them of the direct cerebellar and antero-lateral fibres.

16. The traumatic degeneration, in the sense of Schiefferdecker, is only observed for a space of $\frac{1}{2}$ -1 cm. above and below the section.

17. In the anterior pyramidal tracts of the human brain are fibres which degenerate upward, and which may be separated to form a distinct system (Marie).

MEIROWITZ.

PHYSIOLOGY.

60. A TENTATIVE EXPLANATION OF SOME OF THE PHENOMENA OF INHIBITION ON A HISTO-PHYSIOLOGICAL BASIS, INCLUDING A HYPOTHESIS CONCERNING THE FUNCTION OF THE PYRAMIDAL TRACTS. B. Onuf (State Hospitals' Bulletin, 2, 1897, p. 45).

The author offers the following theory to account for the phenomena of inhibition of the patellar reflexes: "For the excitation of a nerve cell the movement has to pass in the direction from the cell body or its protoplasmic processes toward the nervous process; for the inhibition of the cell the nerve current has to pass in the opposite direction, that is, from the nerve process, or its collaterals, back to the cell body. In other words, to produce excitation of a given cell the nerve current must enter this cell from the surface of its cell body or of its dendrites; but in order to inhibit or moderate the action of the cell the nerve current has to enter the cell from its nerve process or collaterals thereof."

Interpreted in anatomical terms, the author suggests that in the test of the knee jerk the peripheral motor neuron is acted upon in three ways:

1. From the peripheral sensory nerve fibres, probably through a collateral thereof. (The direct reflex arc.)
2. From the cerebellum, which maintains a constant stimulation of the peripheral motor neuron, producing the necessary excitation.
3. From the cortico-spinal fibres, which have an inhibitory action, and thus counteract the other two exciting impulses. "Assuming all this to be the case, interruption of the cortico-spinal pyramidal fibres will give rise to exaggeration of the knee jerk by loss of the inhibitory influence. Interruption of the cerebello-spinal motor tract will cause absolute loss of the reflex, as the sensory stimulus coming from the tendon will be counteracted by the inhibitory action of the pyramidal fibres."

JELLIFFE.

61. LE MÉCANISME DES MOUVEMENTS REFLEXES (Mechanism of Reflex Movements). Van Gehuchten (Neurolog. Centralblatt, 16, 1897, p. 919; abst.).

The author presented this paper at the International Medical Congress of Moscow, an abstract of it appearing in the above mentioned journal.

A number of observers have shown that after complete transverse lesion of the cervical or dorsal region there is a complete loss of the tendon, skin and visceral reflexes, save perhaps the persistence of the plantar reflex upon deep needle puncture. According to our present physiological knowledge these facts are difficult of explanation.

The normal muscular tonus, according to Van Gehuchten, depends upon the condition of excitation which at any moment may be present in the cells of the anterior horns. This excitability depends upon the connecting fibres going to these cells; fibres from the posterior horns and from the cortex and cerebellum through the posterior longitudinal fasciculus.

Such fibres convey either *excitation* (posterior root fibres, intestinal and cerebellar fibres) or *impulses* (cortico-spinal fibres).

The normal muscular tone is thus dependent upon the *nervous* tone of the anterior horn cells, which is imparted to them by these two forms of stimulation, and any change in the nervous tone of these cells affects the muscle tonus. For the consummation of a voluntary movement it is not enough that the fibres from the motor zone to the muscles be intact, the cells in the anterior horns must have their normal nervous tone.

Thus a reflex motor act can occur only when:—

1. The reflex arc is both anatomically and physiologically intact.
2. When the motor cells have a normal nervous tone. Upon this second postulate depends the relationship of the intensity of the reflex act to the peripheral stimulation. Thus it is possible to understand why after the lesions of the pyramidal tracts exaggerated reflexes may be present, without the necessary theoretical assumptions of secondary degeneration or sclerosis of these fibre tracts.

Also it may be explained along some such hypothesis why reflexes may be lost after complete lesion of the cord without calling into account the nervous shock and inhibition fibres, according to Kahler and Pick, or to speak of a functional disturbance of the gray matter of the lumbo-sacral cord.

VOGEL.

62. UEBER DIE LEITUNGSBAHNEN DER REFLEXE IM RÜCKENMARK UND DEN ORT DER REFLEXÜBERTRAGUNG (On the Conduction Paths of the Reflexes in the Spinal Cord and the Locality of the Reflex-Transfer). J. Rosenthal and M. Mendelssohn (Neurolog. Centralblatt, 16, 1897, p. 21).

In contradiction of the accepted view that the short paths be-

tween the ingoing sensory fibres and the outgoing motor fibres are involved in the production of the phenomenon called the reflex, the authors have experimentally shown that in frogs and mammals the reflex-transfer takes place, in the cervical portions of the spinal cord, and perhaps also partly in the medulla oblongata.

After having determined the minimum amount of the irritation just sufficient to produce a definite reflex, sections were made at different levels of the cord and the alterations noted. It was found that the integrity of the connection of the upper cervical cord, immediately beneath the calamus scriptorius, with the sensory and motor paths was indispensable for the production of the reflex. If this region of the cord be destroyed the former effective minimal stimuli no longer produce the reflex. If their strength be increased the stimuli find other paths to the origins of the motor nerves.

It therefore appeared probable to the authors that in cases presenting lesions of the bulbo-cervical region the reflexes below would be either abolished or very much diminished. In diametrical opposition to this view was the teaching that separation of the cord from the medulla occasioned an exaggeration of the reflex.

At that time (18/5) the only reported cases were those of Kadner, Weiss, Kahler and Pick, in which transverse injury in the upper cord caused abolition of the reflexes below the lesion. Since 1882, when the authors renewed their investigations, other cases were put on record in which complete transverse lesion of the cord was followed by flaccid paralysis of the lower extremities with abolition of the tendon, skin and visceral reflexes (Bastian, Bowby, Thorburn, Babinski, Bruns, Gerhardt, Hitzig, Egger, Hoche, Habel, Van Gehuchten), in spite of absolute integrity, microscopically determined, of the lumbosacral regions of the cord.

Another significant fact is that in no case recorded of complete transverse division of the cord did the tendon, visceral and skin reflexes persist.

It appears therefore that the clinical observations are in accord with the experimental in placing the reflex centre in the bulbo cervical portion of the cord.

MEIROWITZ.

63. EXPERIMENTAL OBSERVATIONS ON THE CROSSED ADDUCTOR JERK. P. Stewart (Journal of Physiology, 22, 1897, p. 61).

As the result of the author's observations on a case in which the phenomenon of crossed knee jerks was obtained the following conclusions were offered:

1. That the crossed adductor jerk is not due to direct stretching of the adductor muscles by a shock communicated to the pelvis.

2. That the crossed adductor jerk is not due to a shock mechanically transmitted to the spinal cord.

3. That the crossed adductor jerk is a true reflex, occurring at a period distinctly later than the ordinary knee jerk.

4. That the average time required for the appearance of the crossed adductor jerk is about .126 of a second from the time when the opposite tendon is tapped.

JELLIFFE.

PATHOLOGY.

64. NOTE ON MUSCLE-SPINDLES IN PSEUDO-HYPERTROPHIC PARALYSIS. A. S. Grünbaum (Brain, 20, 1897, p. 365).

The author gives a short note of his findings in a case of pseudo-hypertrophic paralysis. He says the muscle-spindles were for the most part unaffected, but in a few there was a diminution in size of an

intra-fusal fibre with a deposit of hyaline material around. The ordinary muscular, nervous and vascular changes were also present.

JELLIFFE.

65. NOTE SUR LES RÉFLEXES FÉMORAUX CROISÉS CHEZ LES ÉPILEPTIQUES. (On Crossed Femoral Reflexes in Epileptics.) Ch. FÉRET (Comptes rend. Soc. de Biologie, 5, 1898, p. 7).

An examination by the author of some 143 non-paralytic epileptics, examined in a period when but few were deeply under the influence of bromides, revealed the presence of this phenomenon in twenty-four instances. The author presents the following table:—

	Patellar Reflex.	Crossed Femoral Reflex.
Feeble	35	—
Medium	64	8
Strong	44	16
Total	143	24

The variety of the crossed reflex varied; in 16 cases the r. adductor, in 3 the r. extensor and in 3 both muscles showed the phenomena. In general, the crossed femoral reflex was equal on both sides, save in one case, where the right side predominated.

Bromides seemed to have no influence upon either the patellar reflex or crossed femoral reflex.

VOGEL.

CLINICAL NEUROLOGY.

66. LA SCOMPARSA DEL RIFLESSO DEL TENDINE ACHILLEO NELL SCIATICA (Tendon Reflexes in Sciatica). J. Babinski (Gazz. d. Ospedali, 18, 1897, p. 29).

In a note the author shows that, whereas in healthy individuals the tendon-reflex is normal, in sciatica there is usually a diminution or a loss of such tendon-reflex. This phenomenon was found not only in severe cases of the disease, but also in much lighter forms, as ischialgia. In some cases there were marked differences in the reflexes on opposite sides of the body.

The author considers this a valuable diagnostic sign, especially to differentiate simulation from hysterical sciatica. It is not clear, however, that incipient tabes is ruled out in this report.

JELLIFFE.

67. LE RÉFLEXE ROTULIEN DANS LA SYPHILIS (The Knee-Jerk in Syphilis.) Valentine Zaroubine (Jour. de Conn. Méd., March 11, 1897).

The author found the knee-jerk exaggerated in all syphilitic patients during the secondary stage. Following this increase, the excitability often fell and then regained its normal state.

MACALESTER.

68. CASES OF PSEUDO-HYPERTROPHIC PARALYSIS AND OTHER FORMS OF PROGRESSIVE MUSCULAR DYSTROPHY. Bryon Bramwell (Atlas of Clinical Medicine, iii., 1897, p. 95).

In an elaborate report the author presents the clinical histories of fifteen cases of pseudo-hypertrophic paralysis and one case of myopathic muscular atrophy affecting the face, tongue, ocular muscles, muscles of the head, neck, shoulder girdle and upper extremities.

In about five cases detailed microscopical examinations were made of the muscles, the peripheral nerves and the spinal cord.

While there is little new in the communication, the detailed clinical examinations, large number of post-mortems made and the numerous illustrations render this contribution an important one.

JELLIFFE.

69. **ATROPHIA MUSCOLARE PROGRESSIVA SPINALE** (Progressive Spinal Muscular Atrophy). Morasca (Resoconto degli Ospedali di Genova, 1896).

The author describes a case of progressive muscular atrophy of the Aran-Duchenne type, and comes to the general conclusion that if the patient does not come to autopsy, a clinical diagnosis of this disease is alone possible; but that from a pathological point of view the findings may be syringomyelia, or an amyotrophic lateral sclerosis, or an anterior poliomyelitis, a chronic myelitis or a progressive muscular atrophy (Erb). The clinical diagnosis without anatomical confirmation is of relative value only.

VOGEL.

70. **UN CAS D'ATROPHIE MUSCULAIRE PROGRESSIVE DU TYPE DUCHENNE-ARAN.** (A Case of Progressive Muscular Atrophy of the Duchenne-Aran Type). Villers (Journ. Med. de Bruxelles, Jan. 14, 1897).

The case reported here occurred in a man 52 years of age, who five years previously suddenly became paralyzed in the right arm, which recovered in about three months. After this there set in a weakness and gradual atrophy of the muscles of the hand, of the thumb and fingers, which gradually progressed to the arm. One year after his first sudden attack of paralysis he had a peculiar attack of twitching in the left hand, and was taken to a hospital. His condition then showed marked atrophy of the upper extremities, main en griffe, fibrillary twitchings in the opponens pollicis and interossei of the left hand. The muscles of the neck and face were less involved. Sensation and reflexes intact. No reactions of degeneration.

The patient died two years after entering the hospital, having suffered from a number of epileptiform attacks, after each one of which the atrophy became more profound, although his condition had bettered by electrical and massage measures.

Autopsy showed a universal atrophy of the muscles, more profound on the right side. The spinal cord showed a decrease in the size of the anterior horns, the cells being decreased in size. Sections at the level of the anterior end of the optic thalamus showed the position of an old hemorrhage.

Microscopical examination showed general arterio-sclerosis, also involving the brain; degeneration of the muscular substance throughout; in the spinal cord absence of any fibre degenerations, marked atrophy of the ganglion cells of the anterior horns at the level of the cervical and upper dorsal regions. There were no marked changes found in the peripheral nerves, though some showed degenerative lesions.

JELLIFFE.

71. **EXISTE-T-IL UNE ATROPHIE MUSCULAIRE PROGRESSIVE ARAN-DUCHENNE** (Is there a Progressive Muscular Atrophy of the Aran-Duchenne Type?) P. Marie (Revue Neurologique, 5, 1897, p. 686).

In a short note the author discusses the type of progressive muscular atrophy of Duchenne. He states that this was described at a time when our knowledge of the nervous system was quite incomplete, and that subsequent investigations have shown that Duchenne's symptom complex includes a number of diseases, which later writers have been able to differentiate and render classic descriptions of the same.

Some of these have been amyotrophic lateral sclerosis, myopathie progressive primitive, syringomyelia and multiple neuritis.

These considerations lead the author to conclude that there remains no well-defined type to accord with Duchenne's original description, and that the disease, as such, has ceased to exist.

JELLIFFE.

72. WEITERER BEITRAG ZUR LEHRE VON DER HEREDITÄREN PROGRESSIVEN SPINALEN MUSKELATROPHIE IM KINDESALTER, NEBST BEMERKUNGEN UEBER DEN FORTSCHRITENDEN MUSKELSCHWUND IM ALLGEMEINEN (A further Contribution to the Study of the Hereditary, Progressive, Spinal, Muscular Atrophy in Childhood; with Remarks on Progressive Muscular Atrophy in General). J. Hoffmann (Deutsche Zeitschr. f. Nervenheilk., x., 1897, p. 292.

Hoffmann publishes a very important addition to his paper on the spinal form of hereditary muscular atrophy, which appeared in 1893. He reports the case of a child, who was born of sound parents, and in normal labor, and seemed to be healthy until the seventh or eighth month of life. At this time, without known cause and without the signs of an acute or chronic general affection, bilateral, flaccid paresis of the muscles of the hip, buttock and thigh was observed, which gradually extended and involved symmetrically the muscles of the back, neck, shoulder, upper limbs and legs, and caused more or less paralysis of these parts. The muscles first affected were most atrophied. The tendon reflexes were absent. Secondary changes in the joints and vertebral column were noted. Fibrillary tremor was absent. Sensation and the action of the sphincters were normal. The electrical reactions could not be obtained, though reaction of degeneration had been observed in other children of the family who had died with similar symptoms. The mental condition was normal. The facial, lingual and throat muscles were not paralyzed. Death occurred from secondary pulmonary disease when the child was about two and a half years old. The microscopical examination showed symmetrical and marked degeneration of all the peripheral motor neurons below the twelfth nerve, including the eleventh nerve; disappearance or degeneration of the cells of the anterior horns of the cord; great degeneration of the anterior spinal roots; less noticeable changes in the peripheral nerves and the intramuscular nerve branches; distinct degeneration of the crossed and direct pyramidal tracts and of a portion of the lateral column, which was most evident in the upper thoracic and cervical regions, but not traceable above the motor decussation. Simple atrophy in all stages was noted in the muscles. The spinal affection was believed to be primary.

Hoffmann describes a third family in which this hereditary spinal form of atrophy was observed. The diagnostic features of the malady are: the heredity; the commencement in the first year of life; the first signs of atrophic, flaccid paralysis in the muscles of the pelvic girdle and thighs; the extension of the atrophy to the muscles of the trunk and limbs; the absence of tendon reflexes; the presence of reaction of degeneration, and the early death.

Twenty-two cases in all of this disease and four necropsies have been reported. In all four the peripheral neurons were found much degenerated and the muscles in a simple state of atrophy, and in three the anterolateral columns were affected.

At one time only the different forms of muscular atrophy, known as dystrophy, were supposed to be hereditary. Hoffmann speaks of the following four forms of hereditary muscular atrophy:

1. A form which begins in childhood in the muscles of the pelvic

girdle and extends from the trunks to the muscles of the extremities (Werdnig, Hoffmann).

2. An infantile, bulbar, paralytic, facial type (Fazio, Londe).
3. A Duchenne-Aran type (Strümpell, Gowers).
4. A transitional form.

It seems at present that the influence of heredity is more clearly shown in the myopathic and neurotic forms than in the myelopathic form of muscular atrophy. Fibrillary tremor is the rule in the spinal form and the exception in the purely muscular form of atrophy, and yet it may be absent in the spinal form. Reaction of degeneration may occasionally be absent in spinal muscular atrophy and, in exceptional cases, be present in the purely muscular form. Neither simple nor degenerative atrophy of the muscular fibres is a fair criterion of the nature of the atrophy.

SPILLER.

73. BEITRAG ZUR LEHRE VON DER PROGRESSIVEN NEURALEN MUSKEL-ATROPHIE (A Contribution to the Study of Progressive, Neural, Muscular Atrophy). By F. Egger (Archiv für Psychiatrie, 29, 1897, p. 400).

Egger reports two cases of progressive, neural muscular atrophy which occurred in brothers in a healthy family. The disease began in the younger patient when he was thirty-eight. It was first observed in the lower extremities in the peroneal muscles and on the right side of the body. Sensory disturbances were present in both cases. Muscular atrophy and paresis were soon visible in the upper limbs. Reaction of degeneration was also observed. The cases are reported on account of certain peculiar features. In both there was a possibility of poisoning from lead, and it may be that the effects of lead aided in the development of the disease. Vesical disturbance (frequent micturition and dribbling after urination) and swaying of the body on closure of the eyes were unusual symptoms in one patient. One of these cases shows that tabes must sometimes be considered in making a diagnosis of progressive, neural, muscular atrophy, for pain, loss of reflexes, Romberg's sign, girdle sensation and vesical disturbances are certainly very suggestive of tabes. Absence of pupillary rigidity does not necessarily exclude tabes, for an examination of over four hundred cases of tabes in Erb's clinic by Leinbach showed that changes in pupillary reaction were present only in 70.25 per cent., and in cases of one or two years' duration only in 63 per cent. The presence of the disease in two brothers and the early development of the muscular atrophy were contrary to the manifestation of tabes. Egger shares Oppenheim's opinion, viz., that progressive, neural, muscular atrophy is a chronic hereditary form of multiple neurosis; and in this aspect the possibility of lead poisoning in these two cases becomes of unusual interest. Egger thinks with Hoffmann that it is not probable that neuritis may be hereditary, but a slight power of resistance to disease may be transmitted, and it is well in treating a case of progressive neural, muscular atrophy to remove all injurious substances, lead, alcohol, etc., which are so deleterious in neuritis.

SPILLER.

74. BEITRAG ZUR NEURITISCHEN FORM DER PROGRESSIVEN MUSKEL-ATROPHIE (Contributions to the Neural Form of Progressive Muscular Atrophy). Siemerling (Neurologisches Centralblatt, 16, 1897, p. 568, Abst.).

The case described occurred in a young man of 20, whose mother died of tuberculosis. Up to his 5-7th year he developed normally, and then his hands and lower thighs began to atrophy. From his thir-

teenth year he was unable to walk. At first his psyche was normal, but later he became sullen and taciturn.

There was loss of pupillary light reaction, spasmodic twitchings in the left zygomatici, nasal speech with tremor of the tongue, marked atrophy of the hands and arms, main en griffe, the deltoid, pectoralis and biceps being the best preserved; no tendon phenomena, left leg extended, right contracted at the knee; marked atrophy of both sides, loss of active motion in both lower extremities; loss of pain sensations throughout the entire body, more markedly in the legs; fibrillary contractions in the intercostal muscles; marked diminution in the faradic and galvanic currents; hypochondria.

Autopsy showed a normal brain, macroscopic patches in the lateral and posterior columns; fatty degeneration of the lower thigh muscles. Microscopic examination of the cord showed degeneration of the posterior and lateral columns, particularly in the dorsal and lumbar region; atrophy of the anterior horn cells and of Clark's columns; posterior roots intact, anterior degenerated; degeneration of spinal ganglia, sympathetics intact; a widely spread degeneration of the sensory and mixed nerves; degeneration of the muscles in various places. The author believes the peripheral changes to have been the primary ones, the changes in the spinal cord being secondary.

JELLIFFE.

75. BEITRAG ZUR HEMIATROPHIA FACIALIS PROGRESSIVA (Contributions to Progressive Facial Hemiatrophy). J. Donath (Wien. klin. Woch., x., 1897).

The patient described was a man 26 years of age, who ten years previously had received a wound on the right side of the face, which left a scar upon his chin. Three years later the patient had a tooth pulled, and in the operation a portion of the lower jaw was also taken with the tooth. The clinical picture began at that time; the skin was thinned, pale, furrowed; there was an atrophy of the muscles and of the bones, suppression of the perspiration and diminution or suppression of the growth of hair. The sensibility of the right half of the face was diminished and the electrical reactions of the right facialis increased. There also was some atrophy of the right half of the tongue. There was no sensation of pain in the wound, but there was slight pain produced by movements of the lower jaw. The author was of the opinion that the pathological process was due to the infection of the wound at the time when the tooth was pulled, and that a diffuse nerve and vessel inflammation was produced, resulting in the clinical picture described.

VOGEL.

THERAPY.

76. MYOPATHIE PROGRESSIVE AMÉLIORÉE PAR LA MÉDICATION THYROÏDIENNE. (Progressive Muscular Atrophy Improved by Thyroid Therapy). Lepine (Lyon. Medical, 82, 1896, p. 35).

In a case of juvenile muscular atrophy, where the patient was unable to walk or even to stand erect for any length of time, the author gave weekly doses of thyroid gland—120 gms.—with the result that the patient was enabled to stand erect, and finally to go to work. There were no objective changes in the muscles. The author's explanation was that the thyroid secretions might have some influence over the energy of muscle contractility. Suggestion, he believes, was ruled out.

JELLIFFE.

Book Reviews.

A TEXT-BOOK ON MENTAL DISEASES. For the use of students and practitioners of medicine. By Theodore H. Kellogg, A.M., M.D. William Wood & Company, New York, 1897.

The work before us is a large volume of seven hundred and fifty-nine pages. The object of the book, as stated in the preface, is "to set forth in a condensed but comprehensive manner the present state of the science of mental disease. The book is made to embrace the wide range of the history, statistics, nosology, etiology, clinical course, symptomatology, pathology, diagnosis, prognosis and treatment of mental disorders. An attempt has been made to introduce such clear and systematic sub-divisions as would best tend to facilitate the comprehension of the whole subject and render the book available for students and practitioners of medicine. The book aims to be a practical guide to the diagnosis and treatment of all the various types of insanity with which the physicians have to deal in public hospitals or private practice, and also to act as a work of ready reference to psychiatrists in the emergencies of their specialty."

The subject is divided into two parts, the first of which deals with "general mental pathology" and the second with "the special groups and typical forms of insanity." Part first embraces a series of interesting and well-written chapters. Among them are to be specially mentioned one on the nosology of insanity and those of psychical and somatic symptomatology. In that on nosology the subject of classification is considered in a very systematic manner.

However, general statements, generalizations and time-worn platitudes are of little value when compared with the specific statements which should be made regarding the etiology, symptomatology, diagnosis and treatment of each specific mental affection. In the experience of the writer these general sections in treatises of mental diseases are rarely, if ever, read by the student or by the practitioner. What the student and physician need are systematic statements and elaborate clinical descriptions of the actual diseases with which he meets in daily life. It is therefore disappointing to find that, while 516 pages are given up to the consideration of merely general matters, only 241 are given to the actual discussion of the various forms of insanity. The conviction forces itself upon the reader that the special and clinical part of the book must have been condensed and much abbreviated in order to admit of the expansion of the almost useless general section. The writer does not desire to imply that the general section is badly written, but merely that it is disproportionately large, and contains much matter that has no practical application.

Chapter first, on the history of insanity, is sufficiently condensed; that on statistics of insanity had best have been entirely omitted, while that on the nosology of insanity is an admirable review of the various forms of classification at present adopted, but the author errs, in the opinion of the writer, in not contenting himself in the present unfortunate state of our knowledge by simply *enumerating* the various forms in which insanity manifests itself. The chapter on the etiology of insanity could have been much condensed, while that on the evolution, stadia, clinical progression and termination of mental disorders could

with advantage have been entirely omitted. The chapters on psychic and somatic symptomatology are excellent, and deserve unstinted praise, and yet it is an open question whether the student will not gain a clearer conception of the symptomatology of insanity in general by a study of *symptomatology of the specific forms*. In the opinion of the writer this is the only way in which a knowledge of the symptoms is really acquired, especially as insanity is a disorder a knowledge of which can only be acquired by the actual study of living cases. The generalizations present in the general sections of works on insanity are abstractions of clinical experience, and are not materials fit for presentation to the student or practitioner, save to an exceedingly limited degree. The practical value of chapters on the pathology, diagnosis, prognosis and treatment of insanity are also, in the opinion of the writer, equally open to question. Certainly, in the consideration of each specific form of mental disease every factor considered in Part First, from a general standpoint, must be considered a second time in the special section, and it is specific statements of facts in relation to specific affections that are of importance. Considered in this light it is certainly an anomaly that more than two-thirds of the book should be devoted to general considerations and less than one-third to detailed descriptions of the various mental diseases. It is difficult to avoid the inference that as a result of this arrangement the special section has suffered, and an examination of its pages shows this to be the case. The descriptions are for the most part exceedingly condensed. For instance, melancholia is considered in less than six pages; mania in less than eight, while paresis, which is a very world within itself, is disposed of in twenty-two pages, including history, etiology, symptomatology, diagnosis, prognosis and treatment. The other forms of insanity are considered in a like condensed manner.

The author classifies the various forms of insanity into two groups.

Group A "is made in accordance with the etiological and pathological principles, and is hence briefly defined as an etio-pathological and as having assignable etiological and pathological relations." It consists of seven parts: First, insanity from general arrested development; second, insanity emerging from constitutional neuropathic states, usually hereditary, though occasionally acquired; third, insanity with established neuroses; fourth, insanity in connection with the physiological crises; fifth, insanity with general systemic states; sixth, insanity with definite pathological conditions of the encephalo-spinal, vaso-motor or peripheral nervous system, and seventh, insanity from pathological psychic influences.

The second group "consists of the simple psychoses without definitely assignable etiological and pathological relations, and hence the group is briefly named psycho-symptomatological." It consists of the emotional (Class VIII.), the intellectual (Class IX.) and volitional (Class X.) insanities. In the present unsatisfactory state of our knowledge of insanity, almost any classification can be adopted, and a reviewer could reasonably be charged with being captious if he found fault with the classification adopted by any one author. As a matter of fact, the classification, that is to say, the order in which the subjects are considered, is of a relatively slight importance, the real point of importance being the accuracy, the fullness and completeness of the clinical descriptions. A standard work upon insanity should consist of elaborate and comprehensive word-pictures of the things actually met with in the daily life of the physicians in or out of the asylum, and should be interspersed with appropriate records of cases actually studied. This is assuredly not the case in the special section of the work before us.

DERCUM.

SULLA OPPORTUNITÀ ED EFFICACIA DELLA CURA CHIRURGICO-GINECOLOGICO NELLA NEUROSÌ ISTERICA. (E nelle alienazioni mentali.) Risulta di una inchiesta internazionale. Dottori G. Angelucci e A. Pieraccini, Naples, 1897.

As a result of an international inquiry into the relations of gynecology to nervous and mental disorders, and the hopes of operative cure in such cases where such a relationship is stated to be established, the authors present a careful and extended statistical study. In all, they received from alienists, neurologists and gynecologists some 117 replies in full for all the points asked about. As a result of their analysis of these 117 cases, they eliminate at first six cases in which the history would indicate that the results were obtained by suggestion; in the remaining 111 cases it could be shown that in 17 only was there any improvement, the remaining 94 being left in practically the same condition as before. Further, it was to be noted that in the 17 cases 12 would fall under the general head of hysterical disturbance, in 9 of which diseased organs were found, and in only three were these sound. The remaining cases, which resulted favorably, in which the gynecological operations could be said to be responsible, were but five in number; in these two were improved, while three were called cured.

This scarcity and uncertainty of favorable results inclines the authors to believe that in the many reported favorable cases of nervous disturbance cured by means of operations on the pelvic organs another factor is responsible; that, they believe, is suggestion, the operations serving to make the impression more permanent. A brief series of conclusions closes this interesting brochure. These are:—

1. The extirpation of the normal uterus or its adnexa as a means of cure for hysterical neuroses or insanity is distinctly contraindicated.
2. This same condition, "hysteria," even constitutes a contraindication to surgical operations which aim to cure gynecological ailments.
3. Unless there is grave disease of the sexual organs there is no indication for their removal, and any operation for such removal should be considered apart from its remote effect upon the neuro-pathic state.

4. In certain cases in which there are grave pathological changes in the pelvic organs the operative suggestion effects may legitimately be exercised when there are co-existing neuropathic conditions.

Finally, in conditions of hysteria or allied disorders, in which reputable methods of suggestion have proven ineffectual, one may be justified in resorting to a simulated laparotomy. JELLIFFE.

DYSKINESIAS ARSENICAES (Arsenical Dyskinesia.) By Dr. Juliano Moreira, Bahia, 1896. Also abstract in *Revue Neurologique*, 1896, p. 513

This work was a competitive thesis, presented before the Faculty of Medicine and Pharmacy in Bahia.

The first part of the monograph contains eight personal observations of accidental and therapeutical arsenic poisoning by the author. The second part treats of the etiology and toxic symptoms arising from the use of arsenic, studying and dwelling in particular upon impairment of voluntary motility, *i. e.*, *dyskinesia*. After considering and describing in detail the functional disturbances of sensory organs (disturbance of tactile insensibility, vision, taste, sense of heat and pain and muscular sense), the author proceeds to discuss the impairment of the motor apparatus (mechanical and electrical irritability of the affected nerves and muscles). Following these observations a

chapter is devoted to the exhaustive consideration of the pathological anatomy, diagnosis, prognosis and treatment of the conditions, respectively, and an elaborate bibliography is appended.

The treatise shows proof of painstaking, precise and scholarly investigation, and our South American colleague is to be congratulated for having contributed such a valuable addition to our knowledge of arsenical poisoning.

MACALESTER.

DIE DARSTELLUNG KRANKHAFTER GEISTESZUSTAENDE IN SHAKESPEARE'S DRAMEN. Von Dr. Hans Laehr. Stuttgart, Paul Neff Verlag, 1898.

There is hardly any subject in literature which has given rise to more discussion and interpretations than the various cases of insanity in Shakespeare's dramas. It would therefore seem impossible to say anything new on this subject. The little work by Dr. Laehr is nevertheless interesting in various directions, especially as the author endeavors to submit all the different views and theories to a careful investigation. Special chapters are devoted to King Lear, Ophelia, Hamlet and Lady Macbeth, in which the author tries to reach a definite diagnosis in each case. I must confess, however, that any attempt of this kind has always seemed to me of doubtful value, if not quite fruitless. We must not forget that Shakespeare, after all, was not a physician who relates to us the dry history of a case for diagnosis, but that he was the great phenomenal poet, who, true enough, possessed an unusual gift for observation, but who combined this talent with the necessary physical elements of an ingenious artist; *i. e.*, poetical fancy and imagination. To approach his heroes to-day for the purpose of discussing the diagnosis and prognosis of their mental condition from the purely medical standpoint, as we would do at the bedside of our patients, seems to me just as unreasonable as to study the topography of the moon on an oil painting. Whether King Lear suffered from senile dementia, or from paranoia, or, as Dr. Laehr says, from acute confusional insanity, seems to me just as useless to discuss as to criticise the treatment of Lear by his physician, who awakes his patient from sleep with the melodious sounds of music.

Of much greater interest and scientific value to the historical development of psychiatry as well as to the psychological analysis of the ingenious poet are the other questions discussed in Dr. Laehr's work, *i. e.*, "Wherefrom did Shakespeare take the conception of mental diseases?" and "What induced Shakespeare to describe cases of insanity in his dramas?" The chapter devoted to the first question contains a number of interesting data concerning the medical views of insanity at the Elizabethan period, Shakespeare's own observations of psychopathic cases, and the descriptions of mental diseases by poets before Shakespeare. As to the latter question, the motives which induced Shakespeare to picture mental diseases in a dramatic form, I must disagree with Dr. Laehr on several points. His view, for instance that Shakespeare described Hamlet as a psychopathic individual in order to be able to introduce the apparition of the ghost in the tragedy, seems to me to do injustice to the poet. A genius like Shakespeare would never adapt his characters to the surrounding circumstances. The latter would only form the means for expressing and describing the poet's ideas, but could never form the primary motive for a tragedy.

The aims in Shakespeare's dramas are the picturing of the manifold traits of the human character, the conflicts which must necessarily arise from their mutual contact, and the superhuman, divine providence which determines the fate of men, which exercises vengeance

and retaliation. The characters, therefore, were the starting point, to which all other considerations were subordinated. Mental diseases were at that time much more apt to create serious conflicts in public life and the mutual contact of men than nowadays on account of their being recognized less readily, and of the different conception of their origin and nature. The religious doctrine of the independence of the soul from the body and the belief in individual eternal life after death caused, of course, mental aberrations to appear in an entirely different light from bodily diseases. Insanity was the divine vengeance, the tragic punishment for sin and wickedness. That even a genius like Shakespeare stood under such impression seems the more pardonable if we consider that even in comparatively recent times one of the greatest psychiatrists, whose studies of mental diseases became fundamental for modern psychiatry, *i. e.*, *Heinroth*, maintained that sins formed the principal cause for insanity. From this point of view mental diseases must necessarily form one of the most powerful dramatic elements in the tragedy of vengeance. Whether Lear suffered from the one or the other disease is therefore entirely irrelevant. It was neither Shakespeare's intention to describe mental diseases accurately in the medical sense, nor was it possible to do so in the poetical form of the drama.

However much we may differ from Dr. Laehr's views in some respects, his book will be read with pleasure by all who are interested in this subject.

WILLIAM HIRSCH.

DIE AKROMEGALIE. By Dr. Maximilian Sternberg. With 16 illustrations. Published by Alfred Hölder, Vienna, 1897.

This monograph forms part of the large work on special pathology and therapy edited by Nothnagel. The author has carefully searched the literary field of acromegaly, and has put together his findings in a book of 116 pages. The introductory chapter is devoted to the history of the disease. Although the latter was first introduced to the medical world under its present nomenclature by Marie, in 1886, Sternberg tries to show that the affection was known to former investigators under different titles, who, however, never recognized the true position of the malady. Thus as far back as 1772 Saucerotte describes a case with "considerable enlargement of the bones in an adult," which doubtless was an example of acromegaly.

Chapters II. and III. are devoted to the general consideration of the clinical picture presented by acromegaly and to its pathological anatomy. A number of excellent photographs are reproduced, showing the abnormal changes in the features, hands, feet and skull. The hypertrophies in the skeleton appear to be one of the most striking features of the pathological anatomy of acromegaly. Hardly any of the organs have escaped being affected. For the neurologist the most interesting changes are, of course, those in the brain, cord and nerves, and these are discussed at length. Owing to the important role played by the hypophysis in acromegaly an appendix on the morphology and physiology of the hypophysis has been added to the chapter on pathological anatomy.

In Chapter IV. the symptoms of acromegaly are discussed in great detail. This chapter is also illustrated by a number of photographs and by several skiagraphs of the skull and hand.

The chapters following are concerned with the development, course and relation of acromegaly to other diseases. The latter are cranium progenum, myxœdema and cretinism, Basedow's disease, gigantism, diffuse hyperostoses and diabetes. These conditions, as well as a number of others to be presently mentioned, present symptoms which may occur in the course of acromegaly, and hence the

latter affection may easily be overlooked and an erroneous diagnosis made. To prevent such a blunder it is always well to bear in mind the affections for which acromegaly may be mistaken. The author has therefore tabulated them as follows:

A. Diseases whose symptoms participate in acromegaly, and which may be mistaken for the latter:—1. Brain tumors. 2. Basedow's disease. 3. Diabetes. 4. Myopathies. 5. Genital affection. 6. Acroparesthesia. 7. Rheumatism. 8. Traumatic neurosis and its simulation.

B. Diseases and conditions with acromegaly-like alterations in the external appearances:—

I. Anomalies of growth:—1. Gigantism. 2. Congenital partial macrosomia. 3. Congenital and progressive partial macrosomia.

II. General vegetative disturbances (dystrophies):—1. Myxoedema. 2. Cretinism. 3. Basedow's disease. 4. Lymphatic constitution with rachitis.

III. Diseases of the nervous system:—1. Syringomyelia. 2. Erythromelalgia. 3. Various "neurotic hypertrophies." 4. Acquired hemimacrosomia.

IV. Diseases of the bones and joints:—1. Diffuse hyperostosis (megaloccephaly). 2. Multiple tumor-like hyperostosis (Leontiasis ossea). 3. Osteitis deformans Paget. 4. Arthritis deformans. 5. Cranium Progenum. 6. Secondary hyperplastic osteitis (osteoarthropathie hypertrophiante). 7. Multiple symmetrical enchondromata.

V. Diseases of the tendon-sheaths:—1. "Progressive enlargement of hands," Hersmann. 2. Chronic inflammation of the palmar sheaths.

VI. Diseases of the skin:—1. Peculiar pachydermy following prolonged diminution of vessel-tone. 2. Adiposis dolorosa, Dercum. 3. Elephantiasis neuromatodes. 4. Elephantiasis arabum. 5. Oedema.

The treatment of acromegaly is summed up by the author in comparatively few words. As the pathogenesis of acromegaly is unknown, neither prophylactic nor etiological treatment can be instituted.

In addition to dietetic and hygienic measures are mentioned iodide of potash, mercury, thyroid and hypophysis preparations, arsenic and operative procedures. As none of them has been found to be a specific, the therapeutic chapter of the disease is far from being closed.

The bibliography which forms the completing chapter of the work is quite lengthy, containing about 450 references. This is certainly a most valuable feature of the book and will interest those contemplating further studies of the subject.

The book impresses one as being the result of much painstaking labor, and is highly recommended to such as desire to be *au courant* with what has thus far been published on the subject.

PH. MEIROWITZ.

AN EPITOME OF THE HISTORY OF MEDICINE By Roswell Park a.m., m.d., Professor of Surgery in the Medical Department of the University of Buffalo. F. A. Davis Company, 1897.

This epitome of the history of medicine is based upon a series of lectures given by the author at the University of Buffalo. In the preface Dr. Park states what is, we believe, only too true, that "the history of medicine has been sadly neglected in our medical schools. The valuable and fruitful lessons which it tells of what not to do have been completely disregarded."

While the present volume is a little too statistical in its biographic details, it nevertheless is a work that cannot fail to be of value to all

genuinely interested in medicine, past and future. We hope to see it receive a generous recognition. JELLIFFE.

CENTRALBLATT FÜR DIE GRENZGEBIETE DER MEDIZIN UND CHIRURGIE. Edited by Dr. Hermann Schlesinger. Gustav Fischer, Jena, Germany.

This new journal has a very important work to accomplish. In England and in this country specialism has not been carried to the extreme that it has in Germany and Austria, where a physician announces his specialty upon his sign. Even here specialism is rapidly gaining ground. The object of this new journal is to bridge over the gap between the various specialties and to give to the reader a stronger grasp on general medicine. Only abstracts and critical digests on important subjects of the border-line character will be published, and original papers will be left to the recently established journal, *Mitteilungen aus den Grenzgebieten der Medizin und Chirurgie*. The editor is especially well known from his publications on neurological subjects. SPILLER.

SYSTÈME NERVEUX CENTRAL. COUPES HISTOLOGIQUES PHOTOGRAPHIÉES (The Central Nervous System. Histological Sections Photographed). By J. Dagonet. J. B. Baillière et Fils, Paris, 1897.

This photographic atlas is the complement of a paper on general paralysis, published by the writer in the "*Traité des Maladies Mentales*," by H. Dagonet. The pictures have been made from sections of the central nervous system of a patient who was afflicted with the disease under consideration.

Dagonet follows the teaching of Tuzek, and believes that the primary lesions of paralytic dementia are parenchymatous. He disputes the statement made by Weigert in regard to the independence of the neuroglial cells and fibres. He believes also that the pericellular spaces are not artifacts. The atrophy of nerve cells and their fibres, as well as the hypertrophy of the neuroglial cells and their fibres, is well shown, and the hyaloid bodies, which the writer says have received little attention, are described. The latter have been recently studied by Edsall and Sailer, and the reviewer has found them in large quantities in a case of basal tumor of the brain.

Dagonet does not believe that the ependymal granulations, so commonly seen in cases of general paralysis, are caused by primary destruction of the ependymal epithelium (Weigert) and proliferation of the unrestrained neuroglia. When the ependymal cells proliferate in paralytic dementia their fibres, which are very visible in the embryo, but not in the adult, become thickened and are easily perceived. There is, therefore, a return to embryonic conditions, and the greater part of these granulations is formed by the ependymal and neuroglial fibres. Dagonet has not been able to observe the mitosis in the ependymal cells which has been seen in the embryo. He describes changes in the motor cells of the cord, but not of so intense a degree as those of which Berger has recently spoken.

The book is short, and only contains twelve plates, but it is written in an interesting style. SPILLER.

A year-book, with the title "*Jahresbericht über die Leistungen und Fortschritte auf dem Gebiete der Neurologie und Psychiatrie*," will be published under the direction of Mendel, Flatau and Jacobsohn. The first number will appear in 1898. As the various departments will be under the charge of well-known specialists, the book will doubtless prove of great value. The publisher will be S. Karger, in Berlin.

BOOKS RECEIVED.

"A Manual of Legal Medicine," by Justin Herold, M. D. J. B. Lippincott Co., Philadelphia.

"The American Year Book of Medicine and Surgery," edited by Geo. M. Gould, M. D. W. B. Sanders, Philadelphia

"Orthopedic Surgery," by Jas. E. Moore, M. D. W. B. Sanders, Philadelphia.

The Report of the Board of Health of the City of New York to Hon. Wm. L. Strong, entitled "Pulmonary Tuberculosis."

"Preventive Medicine in the City of New York," address in public medicine, Hermann M. Biggs, M.D., Health Dept., New York City.

"The American System of Practical Medicine," Vol. iii. Edited by Alfred L. Loomis, M. D., and Wm. Gilman Thompson, M. D. Lea Bros. & Co., Philadelphia.

A letter from Dr. Bechterew:—

It is proposed to establish two museums, a psychiatric and a neurological, in commemoration of the thirtieth anniversary of the founding of the psychiatric clinic in St. Petersburg.

In the psychiatric museum there will be collected:

1. Plans, photographs, models, etc., of psychiatric institutions, etc.

2. Models of rooms, dress, etc., of the insane.

3. Work done by the insane.

4. Photographs of the insane at different periods of their disease, etc.

5. The apparatus used in examining the insane.

6. Skulls and brains of the insane and microscopical preparations.

7. Documents relating to the care of the insane.

In the neurological museum there will be collected:

1. Brains of animals and men and microscopical preparations.

2. Preparations of pathological brains.

3. Instruments for weighing and methods of preserving the brain.

4. Photographs and representations of pathological processes.

5. The different forms of apparatus employed in treatment.

6. Plans and photographs of neurological institutions.

The museums are intended for the use of students of all countries, and assistance is asked in carrying out the above plans. Plans of buildings, samples of the work of patients, statistics relating to institutions, signed photographs of physicians connected with institutions, anatomico-pathological preparations, etc., are desired.

The expenses of transportation will be lessened if the articles are addressed: Russie, St. Petersburg. Clinique des Maladies Mentales et Nerveuses, Rue Samarskaya, No. 9.



DR. EDWARD CONSTANT SEQUIN.

THE
Journal
OF
Nervous and Mental Disease

Obituary.

EDWARD CONSTANT SEGUIN, M. D.,
New York.

Dr. Edward C. Seguin, the distinguished neurologist of this city, died on Saturday evening, February 19th. His health began to fail in the winter of 1894-'95; but, in spite of progressive loss of strength, he did not give up professional work until July, 1896. Soon after this time the real nature of his illness became apparent, and from this time he was confined to his home, growing gradually weaker, but retaining his great mental powers almost to the last.

Dr. Edward Constant Seguin was born in Paris, France, in 1843. He was the only child of Dr. Edward O. Seguin, whose brother, father and several relatives of the same name were physicians, chemists, engineers and architects. Dr. Edward O. Seguin devoted nearly all his life to the training and education of idiotic and backward children, in France and in this country. He was the originator of the "physiological method" of education, which method was based (as far back as 1873-'78) upon the principle of training the special senses and the two hands (muscular sense) as the means of developing the cerebral functions. It included in its practical details most of the work now known as "object-lessons" and "kindergarten" drill. In 1850 Dr. Seguin, foreseeing the inevitable success of the policy which culminated in the bloody coup-d'etat of December 2d, 1851, emigrated to this country with his family, and finally settled in Cleveland, Ohio.

There, and in Portsmouth, Ohio, the subject of our sketch received a good public and high school education. One year of this time was given to an apprenticeship at the wheelwright trade in Portsmouth. Circumstances made it impossible for him to go to college. In 1861, then residing at Mt. Vernon, New York, he began the study of medicine with his father, attended three courses of lectures at the College of Physicians and Surgeons, New York (the Medical Department of Columbia University), and was graduated therefrom in the autumn of 1864. Meanwhile, from May, 1862, Dr. Seguin had entered the medical department of the army, serving for the first two months (when less than nineteen years old) as "dresser" in the hospital steamships of the U. S. Sanitary Commission, in the Pamunkey and James Rivers. In July he was appointed a Medical Cadet in the regular army, and served two terms, till August, 1864. During much of this time he had, practically, the charge of the patients in the wards to which he was attached, performing all the duties of surgeon, except the doing of major operations. In this service, living in the hospitals, he developed non-tubercular phthisis in the spring of 1864, from the effects of which he did not recover for several years. From September, 1864, to June, 1865, he served at Little Rock, Ark., as Acting Assistant Surgeon, and during the last two months as Assistant Surgeon, U. S. Volunteers. From 1865 to 1867 he passed through the grades of interne and house physician in the New York Hospital, then at Broadway and Duane street. Early in 1868 symptoms of phthisis reappeared, and he applied for a position in the medical department of the army. By special courtesy of the Surgeon-General he was assigned to duty in New Mexico, and there served as post surgeon at Forts Craig and Selden. In the summer of 1869 he returned to New York entirely cured (as the result showed) of his pulmonary trouble.

The winter of 1869-'70 was spent by Dr. Seguin in

Paris, studying privately under Brown-Sequard, Charcot, Ranvier and Cornil, masters whose friendship he always retained. This course of study led him to look forward to making nervous diseases a specialty; but after his return to New York he entered upon general practice in association with Dr. William H. Draper. In 1876 this friendly association was severed, in order that he might devote himself exclusively to the study and treatment of nervous diseases. From 1871 to 1885 Dr. Seguin was connected with the Faculty of the College of Physicians and Surgeons, lecturing upon diseases of the spinal cord and upon insanity. In 1873, with the permission of the Faculty, he founded the Clinic for Nervous Diseases, which, though unavoidably placed upon an unfavorable day (*viz.*, Saturday afternoon), prospered satisfactorily. From 1882 to 1893 Dr. Seguin was in Europe several times, but resumed the practice of his specialty whenever he was in New York.

Dr. Seguin has written many monographs relating to nervous diseases, more especially to their treatment by hygienic as well as by medicinal means, and a number of these were edited in book form, entitled "*Opera Minora.*" Various circumstances, and the belief that there were already too many books upon the subject, prevented him from carrying out a long-cherished plan of writing a formal treatise upon nervous diseases.

He was one of the founders of the American Neurological Association and of the New York Neurological Society, and these, with the New York Pathological Society, received most of his attention. He was also a member of the New York County Medical Society, Academy of Medicine, besides several European medical societies.

Although naturally disposed to the scientific study of medicine, it was always his guiding principle to make everything subservient to the welfare of each patient who intrusted himself to his care. The chief objects of medicine he believed to be the cure, alleviation and prevention of disease.

The Neurological Society records with profound sorrow the death of Dr. Edward Constant Seguin, who died of cirrhosis of the liver on Saturday, February 19th, 1898, aged 54 years.

Dr. Seguin was one of the founders of this society, and its president during 1877-'78, and was for many years a constant attendant at its meetings, taking a leading part in its scientific discussions; his opinions being always listened to with that respect to which his vast clinical experience and his sound judgment entitled them.

For many years Dr. Seguin ranked with the foremost neurologists of the world, and his contributions to science were valued highly.

Whereas, This society has lost by the death of Dr. Seguin one of its original incorporators and an earnest supporter; therefore be it

Resolved, That in his death this society has lost a valuable and honored member. In his professional attainments he was most eminent; enthusiastic in his devotion to his special field of work, in which he was justly esteemed an authority. As an author he was remarkable for his acute observation and logical reasoning, while the clearness of his style gave evidence of the directness of his thought. By his death scientific neurology has lost a zealous and successful disciple, while the community has been deprived of the services of a skillful counsellor and practitioner. The members of the Neurological Society desire thus to give expression to their feelings of respect for his memory and, sympathizing sincerely with his family in their loss, offer them respectful condolence;

Resolved, That this minute be entered upon the records of the society and that a copy be sent to the family of Dr. Seguin and to the medical journals.

J. ARTHUR BOOTH, M.D.,
M. ALLEN STARR, M.D.,
GEORGE W. JACOBY, M.D.,

Committee.

Original Articles.

SEXUAL PERVERSION OR VICE? A PATHOLOGICAL AND THERAPEUTIC INQUIRY.¹

BY MORTON PRINCE, M.D.,

Instructor in Diseases of the Nervous System, Harvard Medical School.

It is not necessary for me before a society of experts in psychiatry to dwell upon the medical, social and forensic importance of sexual perversion. Nor need I, for the purposes of this paper, more than mention the different ways in which the sexual instinct may be perverted. It may be excited by, and therefore lead to acts of cruelty or violence inflicted upon, the opposite sex (Sadism), or by the opposite state, the passive suffering of pain which has been inflicted by the opposite sex (Masochism), or it may be excited by certain objects, whether a part of the female body or dress, or other objects (Fetichism).

Perversion may further take the form of homo-sexuality, that is, the substitution or co-existence of sexual feeling for the same sex in place of, or by the side of, that for the opposite sex. This is also known as *contrary sexual instinct*, or *sexual inversion*. These different forms of perversion have also been classed as varieties of sexual paræsthesia.

The object of this paper is not to present any new facts or new theories of these aberrations, but rather to re-examine the grounds on which the later and dominating views of the nature of these so-called perversions are based, with a view of ascertaining whether these views are really well founded, and whether, after all, the facts

¹ Read before the Medico-Psychological Society, Jan. 27th, 1898. This paper is largely based on the article on "Sexual Psychoses" by the writer in vol. iv. of the Amer. Syst. of Practical Medicine, now in press.

upon which they are supposed to rest have been so well proven that we are justified in accepting the prevailing doctrine. Further, it may be inquired whether the common acceptance of those views which marks most of the contemporary literature is not due to the influence of personal authority rather than a careful consideration of the facts. We may also inquire whether, by taking a broader view of these anomalies, we may not class them with many well-recognized non-sexual manifestations of hysteria, neurasthenia, or other forms of nervous degeneration, and find a common method of treatment.

As the time limit of such a paper does not admit of a separate study of each of the perversities, I shall limit myself, for the most part, to an examination of the one which is most frequent and has received the widest discussion, namely, contrary sexuality, prefacing this special inquiry by a few general remarks on the perversities as a whole.

The first important question is: How far are these perversions the necessary expression of a disordered nervous system, and how far do they represent merely indulgences in vice and cultivated habits? So far as they are simply vicious habits, they can only be regarded as *perversity*, not *perversion*; that is, as vice, not disease. This view is not altered even in the case of individuals who have degenerate or in other ways diseased nervous systems, provided that they have cultivated the habits, and that the habits are the direct result of such cultivation. A paranoiac or an imbecile may cultivate vice as well as sound-minded people. Degenerate people may not be morally or legally responsible; but this inquiry is not one of responsibility, but of *genesis*. What is the origin of and what influences have developed the sexual aberrations? If these are the manifestation of a diseased nervous system, in the same sense as hysteria is the manifestation of a neuropathic condition, then these sexual phenomena are true perversions and pathological.

On the other hand—this point may further be insisted upon, in addition to what has just been said—the presence of a psychopathy does not necessarily indicate that the sexual habits are not vice. A person who, let us say, has by inheritance received a neuropathic constitution, or even exhibits weakmindedness or some form of insanity, is no less subject to the same influences as are normal individuals. His nervous system is equally open to education; so that feelings, tastes and habits can be cultivated in the one as in the other. But it cannot necessarily be inferred from the co-existence of such habits and a psychopathy that the former is the symptomatic manifestation of the latter. Hysterics, degenerates and imbeciles have less intellectual power for resisting feelings and external influences than healthy people. Their judgments are warped, and they do not normally foresee the consequences of their actions, or, if they do, they are not influenced thereby in their conduct. They may not have a normal power of self-control, of restraining impulses. From this lack of resisting power they are more likely to submit to external influences and cultivate the gratification of their senses; that is, they are more likely to be the victims of vice than normal persons. But a diminished resisting power does not make the thing to be resisted pathological; for this other conditions are necessary.

The power of resistance is a force of varying quantity in different individuals, and the difference between the resisting power of a normal individual and a diseased one is only one of degree, and may be small.² Nevertheless, though such diseased persons are more likely to exhibit vice than healthy people, it is, a priori, possible that what would be vice in others may be only a pathological phenomenon in them. Feelings and actions may be only

² What degree of lack of resisting power constitutes *irresponsibility* is still another question, and a distinct one, of an entirely different nature, which does not change the nature of an act, otherwise vicious, but may affect the culpability.

pathological and necessary reactions of a diseased nervous system to external influences. A careful inquiry is, therefore, essential to determine which of these conditions obtains.

These observations are here made, because there is considerable and important difference of opinion regarding the pathology and nature of these sexual aberrations, and, as it seems to the writer, much confusion of thought regarding their relation to disease and vice.

There are two views regarding the nature of perversion which are radically opposed, and which, from a social and therapeutic point of view, have respectively important consequences. The one leads to therapeutic nihilism and social hopelessness; the other offers hope and possibilities.

The theory that has been most widely accepted by writers on the subject is that sexual perversion has its basis in a diseased nervous system, which, in most cases, is the result of inheritance. A psychopathic or neuropathic groundwork is in almost all cases essential, but the perverse phenomenon arises spontaneously, without external cause. Its origin is, therefore, entirely independent of cultivation by vicious habits, education or seduction. In some instances it is equally maintained that these perversions are *acquired* as the result of cultivation, with or without the co-operation of an inherited neuropathic condition. But it would seem that, with the exception of fetichism, which is always acquired, according to this school, the acquired cases are a distinct minority. In most cases *nasçitur, non fit*.

"This perverse sexuality," says von Krafft-Ebing, speaking of the contrary sexual instinct, "appears spontaneously, without external cause, with the development of sexual life, as an individual manifestation of an abnormal form of the *vita sexualis*, and then has the form of a *congenital* phenomenon; or it develops upon a sexuality, the beginning of which was normal, as a result of any definite injurious influences, and then appears as an acquired

anomaly. . . . Careful examination of the so-called acquired cases makes it probable that the predisposition also present here consists of a latent homo-sexuality, or, at least, bi-sexuality, which, for its manifestation, requires the influence of accidental causes to rouse it from its slumber."³ While objections may be made to this theory when applied to homo-sexuality, as has been done with great force by von Schrenck-Notzing,⁴ the theory has considerable strength when we seek for an explanation of Sadism and Masochism. Between the homo-sexual influences and the sadistic influences, which lead to murder and mutilation of the victim's body, there is a wide gulf, and we should not necessarily expect a similar pathological condition as a basis of both. As to Sadism, von Krafft-Ebing expresses the opinion that, "as a rule, it may be safely assumed that the psychopathic state (perverse instinct) exists *ab origine*."

"Sadism is, then, nothing else than an excessive and monstrous pathological intensification of phenomena—possible, too, in normal conditions in rudimentary forms—which accompany the psychical *vita sexualis*, particularly in males."⁵ The same writer lays stress on the weakness or absence of all restraining ideas in the psychopath who gives free hand to the development and expression of the congenital perversion. But he neglects the influence which a deliberate cultivation may have upon a mild impulse or sensory association at the beginning. If Sadism is an "excessive and monstrous intensification of phenomena" existing "in a rudimentary form" in *normal* individuals, then the perversion is the *intensification*, and

³ "Psychopathia Sexualis" (p. 187), translated by Charles Gilbert Chaddock, M. D., 1893. See also "Zur Erklärung der conträren Sexual-empfindung," *Jahrbücher für Psychiatrie und Neurologie*, 1895, by the same writer.

⁴ "Suggestive Therapeutics in Psychopathia Sexualis." Translated by Charles Gilbert Chaddock. 1895.

⁵ "Psychopathia Sexualis," p. 60.

the question is, To what is this intensification due? Does it exist, *ab origine*, in its intense form as a result of pathological development, or is it a later disease symptom, or is the intensification due to cultivation by a morally depraved and mentally weakened individual? Or, may it be due to two or more of these factors? The autobiographies and histories of cases found in the literature do not allow of the first interpretation. It is possible that certain anomalous sensory associations may be the starting point of such perversion, and cultivation does the rest. For example, the case was brought to my attention of a perfectly healthy, mentally and physically, medical man who was sexually excited by the sight of a surgical operation. This person is a typically strong and healthy-minded man. Suppose him to have been a mental degenerate; how easy it would have been for him to cultivate sadistic impulses. The origin of sadistic impulses is of less practical importance than is that of contrary sexuality, as most of the individuals who exhibit them are otherwise psychopathic (e. g., imbeciles, degenerates or insane), though the question is of some importance forensically as bearing on the question of responsibility.

Von Krafft-Ebing's⁶ work being almost the first to treat systematically the subject of sexual perversion, and presenting the matter with great erudition, has been very widely drawn upon by subsequent writers. The interpretation of these aberrations given by the author has very profoundly influenced medical opinion, and has been quite extensively accepted. This work was soon followed by a publication on the contrary sexual instinct by A. Moll,⁷ who also adopted the congenital theory, originally proposed, it is true, by Casper,⁸ in 1852. Quite a large num-

⁶ According to von Krafft-Ebing, the most important previous writings were those of Moreau ("Des aberrations du sens générique") and Tarnowski ("Die krankhaften Erscheinungen des Geschlechtes-sinnes.").

⁷ "Die conträre Sexualempfindung." Berlin, 1891.

⁸ Westphall adopted the congenital theory for contrary sexuality.

ber of contributions to the subject, with reports of numerous cases of different kinds of perversion, have appeared since von Krafft-Ebing's work. In America, Kiernan, Chaddock and Lydston have advocated the congenital theory. More lately, a strong protest against these views has appeared in the work of von Schrenck-Notzing.⁹ This author, in opposition to the opinion of the writers just cited and of others, has urged with great force that sexual perversion, instead of being an original psychopathy, is a cultivated instinct. Heredity and a neuropathic constitution play an important part, but this part is only that of weakened power of resistance to external influences. The contrary sexual instinct is, as such, not inherited, nor is it congenital any more than are the majority of psychoses, but only that tainted or degenerated nervous system, in consequence of which the individual offers a mental weakness, a lack of resistive power to external influences and a lack of control over desires, however excited. By a process of cultivation the neuropath develops feelings and gives them expression in outward acts, over which he sooner or later may lose all control. The first awakening of the perverse instinct may be entirely fortuitous, or by auto-suggestion, or it may be seduction, or other accidental external circumstances; from this time on it is a process of education. Von Schrenck-Notzing would explain in this way the origin of all forms of sexual perversion, although in the exposition of his theory his argument is devoted almost entirely to the contrary sexual instinct.

The influence of von Krafft-Ebing's able exposition of the subject, as just said, has colored much of the writings of others. But I think the conviction must be forced upon the careful student of these writings that the attempt to make vicious habits the result of congenital anomalies has been based upon evidence that, from its very nature, must be incomplete and unreliable.

⁹ "Suggestive Therapeutics in Psychopathia Sexualis." Translated by Charles Gilbert Chaddock, M. D., 1895.

In support of this statement let us consider briefly, but with more particularity, the pathogenesis of contrary sexuality, in accordance with the design mentioned at the outset.

This aberration, as has been said, consists in the existence of sexual feeling for the same sex, co-existing in its fully developed form, with entire absence of sexual feeling for the opposite sex. In the more moderate form there may still be inclination toward the opposite sex, but in the higher degrees of the perversion there may be a feeling of actual repulsion for the opposite sex, while the whole psychical personality, the tastes, feelings and modes of thought of the individual may become changed to correspond with the sexual perversion; that is, the character of the male becomes feminine, and vice versa. Now, the thesis is that all this change of character, psychical as well as sexual, is not only congenital, but a partial manifestation of a neuropsychopathic state, in most cases hereditary. Hence it is in no sense a perversity or vice, but a true anomaly or perversion of instinct, in the sense that it is the product of mal-development, in the same way that any of the normal instincts, tastes or sensory functions are the product of normal development. In other words, with "a normal anatomical and physiological state of the (genital) organs a sexual instinct may be developed which is the exact opposite of that characteristic of the sex to which the individual belongs." It appears spontaneously, without external cause, with the development of sexual life.

Various theories have been proposed, many of them fanciful, to account for the development of this (according to this view) anomalous condition. But, as all these theories presuppose the congenital character of the anomaly, a serious consideration of them is hardly in order until the phenomenon has been proved to be congenital.

Nevertheless, a brief statement of some of their theories makes the point of view of these writers more

definite. Ulrichs, himself a pervert, thought that a female mind was inclosed in a male body; that is, that there were people, to whom he gave the name of "Urnings," whose bodily structure was that of a man, whose sexual instincts were those of a woman. He also considered this condition due to atavism.

This same idea has been put in a more taking form by Kiernan,¹⁰ who rests it upon the biological facts that bi-sexuality, or hermaphroditism, is found in the lowest orders of life, and that in the human embryo this same bi-sexuality exists up to a certain age. In degenerates there is a "throwing back," to use the language of the kennel, to this primitive embryological form, to the extent that, while in the adult male there is a differentiation of anatomical form (sexual organs), the nervous system is developed on the female type. To quote his language, "The original bi-sexuality of the ancestors of the race, shown in the rudimentary female organs of the males, could not fail to occasion functional, if not organic, reversion, when mental or physical manifestations were interfered with by disease or congenital defect." Again, . . . "It seems certain that a femininely functioning brain can occupy a male body, and vice versa."

The lowest animals are bi-sexual, and the various types of hermaphroditism are more or less complete reversions to the ancestral type. Lydston¹¹ advocates the same hypothesis, which is meant to be the principle of atavism. I say, meant to be, for it seems, oddly enough, to have been overlooked that there is very little atavism about it. If there was a development of a female anatomical structure we might talk of "throwing back," but when the anomaly consists, as it does, of psychical phenomena and nervous reflexes, it is difficult to see how there can be a reversion to or inheritance of a nervous system which never had any

¹⁰ Med. Standard. Nov., 1888.

¹¹ Med. and Surg. Reporter. Sept., 1889.

existence in the lower order of life, and never came into existence until the present mono-sexual being was evolved. It is only fair to state that credit may be given, if credit is desired, to one of v. Krafft-Ebing's patients, by whom this theory was proposed.

Chevalier has proposed a modification of this theory in this wise: Originally, in the embryo, man is bisexual; in the future development there is a sort of contest, in which one or the other factor, male or female, develops at the expense of the other, with the result of a mono-sexual individual. But traces of the other sexuality remain. Under certain conditions these latent sexual characteristics successfully succeed in developing themselves, and contrary sexuality results.

Magnan and Gley also imagine a female brain in a male body.

In all these expositions there is a naïve assumption that in the brain of either sex there is a sort of nervous mechanism, peculiar to either sex, and corresponding to the difference in anatomical form—a hazy sort of cerebral localization involving a different cerebral architecture for each sex. This seems to me nothing more nor less than the old-fashioned phrenology, and difficult to reconcile with the first principles of psychology.

Westphal, who first gave the name contrary sexuality, thought the condition was congenital, but refrained from hypothesis, excepting that in one case, in which there were olfactory hallucinations, he thought this particular phenomenon was atavistic from the lower animals. Westphal, who also regarded the condition as a psychosis, gave the impetus to the publication of a long list of cases by other writers.

The best congenital hypothesis is undoubtedly that of von Krafft-Ebing, who thinks that an explanation "may perhaps be found in the fact that it represents a peculiarity bred in descendents, but arising in ancestry. The hereditary factor might be an *acquired* abnormal inclination for

the same sex in the ancestors, which, being transmitted, becomes fixed as a congenital, abnormal manifestation in the descendents."

Kiernan had also suggested this possibility for certain cases.

On the other hand, Binet considers that the whole perversion is acquired through the force of the law of association of ideas.

Among the names of those contributing to the subject are to be found many of well-known writers in neurology and psychiatry. But the most important contributions are those already mentioned, and particularly the works of Moll,¹² von Krafft-Ebing¹³ and von Schrenck-Notzing.¹⁴ The difference in the views of these writers has already been pointed out. Besides the fact that the manifestations of contrary sexuality are acquired, von Schrenck-Notzing holds that nevertheless these manifestations become in time imperative sensations and imperative ideas, and thus from this point of view may be looked upon as psychoses—artificially created in a neuropathic soil in most instances. This opens a very wide field for discussion, as it is no easy matter to settle what decisive element constitutes an imperative idea. The familiar language of the pervert, which is stereotyped in "irresistible impulse," too often should be written, "I don't want to."¹⁵ Still, we must allow, as we see in the alcoholic and opium habit, that for weakened resisting powers sensations may be well educated to such an extent as to become imperative.

¹² "Conträre Sexualempfindung."

¹³ "Psychopathie Sexualis" and "Zur Erklärung der conträren Sexualempfindung."

¹⁴ "Suggestive Therapeutics in Psychopathia Sexualis."

¹⁵ "I wish to state expressly that, though I am conscious of the abnormality of my inclinations, I have no desire to change them; I, long only for a time when more easily, and with less danger of discovery, I can give rein to my desires and experience a delight that will harm no one."—Autobiography; case 149, v. K.-E.

Now, putting aside hypotheses of the "how," an examination of the congenital perversion theory shows that it rests entirely upon the autobiographies of perverts, and certain assumptions (to be presently mentioned) regarding the normal development of the *vita sexualis*, and of the tastes, habits and modes of thought peculiar to each sex.

It is believed that a person is capable of remembering all the circumstances attending the gradual growth of the sexual functions in early childhood, has a distinct recollection of the causes which first called it forth, and that a failure to remember possible excitants is equivalent to their non-existence. A reliance upon evidence of this kind in any other department of human knowledge, whether medical or non-medical, I am sure, would only excite surprise. Even in taking an ordinary medical history, we should hesitate to accept such testimony as final, and I think we should be even more cautious in our examination of autobiographies which attempt to give an analysis, founded on introspection, of the feelings, passions and tastes of degenerate individuals who attempt to explain their first beginnings in early childhood, and attribute each to its proper excitant. As von Schrenck-Notzing has pointed out, in his very careful study of the published cases, very few of these autobiographies will stand analysis. Probably there is no class of people whose statements will less stand the test of a searching cross-examination than the moral pervert. One cannot help feeling that if the pervert was thus examined by an independent observer, instead of being allowed to tell his own story without interruption, a different tale would be told, or great gaps would be found, which are now nicely bridged, or many asserted facts would be resolved into pure inferences.

Taking one point alone, it is extremely doubtful whether any one can remember the first beginnings of the *vita sexualis*. He may remember certain occasions, which, from the special intensity of the excitation, or from peculiar associations, persist as vivid mental pictures; just

as we remember certain pleasurable experiences of boyhood connected with sports, but not all nor the first.

The second error of those who maintain the congenital theory is that they overlook the influence which casual external circumstances have in suggesting feelings and ideas to the mind, and in directing thoughts which appear to be spontaneous.¹⁶ These external circumstances may be trivial or not, and may be forgotten. Even when very prominent for the moment in consciousness, they may be forgotten, while the effects may persist. The enlargement of our knowledge of the substrata of consciousness, and the after-influence of such subconscious states upon the personality of the individual, has made it possible for us to understand the genesis of certain neuroses which before were inexplicable. Janet has demonstrated this influence in the production of some of the manifestations of hysteria. With this knowledge it is next to impossible to say that sexual aberrations were not originally suggested by external conditions in individual cases, or the product of auto-suggestion.¹⁷ A very suggestive example of the influence of this kind upon the lower strata of consciousness in producing psychoses is the following, from the writer's own experience: A young girl, about 16 years old, was pursued with an uncontrollable fear of vomiting. As a matter of fact, she never did vomit, but the fear was so intense that she was unwilling to leave the house alone, or,

¹⁶ Whether or not a neuropathic taint is necessary, as has been maintained, is a secondary matter. The existence of an hereditary taint has, however, been sometimes accepted on insufficient evidence.

¹⁷ A capital illustration of the influence of forgotten causes in producing physical phenomena is the following: A lady told me of a dream, in which she saw distinctly the face of a person whom she had never seen. Her description of the person being very accurate, I insisted, to test the matter, that she must have seen or heard of the person before. On assuring me of the impossibility of this, I told her, as was the fact, that a few days previously I had described this person to her, using the *same language* that she now used for the same description. She had no recollection of it. Sexual suggestions and excitants might be similarly forgotten.

for that matter, even when accompanied, go in places of amusement, or to such distances from home that she could not quickly reach her house. The fear, although always present, was subject to exacerbations. In such attacks her suffering was very great and the mental state uncontrollable. She would take off her clothes, and run up and down the room, crying and begging her mother not to let her vomit. This fear had apparently developed spontaneously during early girlhood, and might easily have been considered congenital if the original history, as given by the patient herself and mother, had been believed. But from the mother, after persistent inquiry, I obtained the following history, till that moment forgotten: When the patient was a child, *sáy*, 5 years old, her sister was taken ill with scarlet fever, the first symptom of which was violent vomiting. That the other child (my patient) might be prevented from catching the disease, she was told that if she went near her sister she would be taken with vomiting in the same way. This had the desired effect, but when the sister recovered it was with some difficulty that my patient could be induced to come into her presence. She ran away and hid in a closet, exhibiting considerable fear. It is reasonable to suppose that the impression made upon the mind at that time had left a subconscious idea, which was the cause of the apparently motiveless fear later exhibited. The patient had no memory of all this. The excitation of abnormal sexual feelings may well have similar external causes long since forgotten.

The third error of this school is that it assumes that normally there is a hard and sharp line drawn by nature between the normal personalities of the sexes. As a matter of fact, sharp lines of demarcation do not occur any more than in the length of the nose or size of the hand. Taking a large number of people, the male personality normally shades into the female, and vice versa. What I mean to say is that, taking a large number of normal males and an equal number of normal females, we might

place them in a row, so that at one end would come the males with strong, vigorous, masculine characters; in the middle, but at the extreme end of the male line, the men with female personalities; adjoining these the masculine females, differing but slightly except in anatomical form from the males; while at the extreme end of the female line would come those with strongly marked feminine personalities.

Fourthly, the effect of education, meaning by this the total environment, intentional education, unconscious mimicry, external suggestion, example, etc., etc.—the effect of this, I repeat, in moulding the tastes and habits of thought and manners of the child, and thus differentiating those of one sex from those of the other, has been overlooked.

I think it is extremely probable that if a boy were brought up as a girl and a girl as a boy, and absolutely freed from all counter influences, such as the unconscious influence of public criticism, etc., each would have the non-sexual tastes and manners of the other sex.

Fifthly, it is questionable whether only abnormally the *vita sexualis* of the male is excited by the female, and conversely. There is every reason to believe that in some perfectly healthy individuals some degree of erotic feeling or ideas may be excited by the sight or touch of the form of a person of the same sex, and, at any rate, thoughts (pertaining to anatomy) so excited may very naturally awaken secondarily associated sexual feelings. For instance, the *vita sexualis* in a boy is first associated with his own sexual organs. Later, the sight of those of another boy awakens the association of ideas by the well-known law, and then, in a degenerate, cultivation does the rest. Von Krafft-Ebing's first case (106) of a girl with hyperesthesia sexualis and homo-sexuality is readily explainable in this way. As von Krafft-Ebing points out, in the beginning of sexual development in the child "the psychical relation to persons of the opposite sex is still

absolutely wanting, and the sexual acts during this period partake more or less of a reflex spinal nature." "With the inception of anatomical and functional development of the generative organs, and the differentiation of form belonging to each sex, which goes hand in hand with it in the boy or girl, rudiments of a mental feeling corresponding with the sex are developed, and in this, of course, education and external influences in general have a powerful effect upon the individual, who is now all attention." Now, in a person of perfectly healthy mind and body, all social customs, habits of thought, unwritten laws, and moral precepts tend to suppress any existing homosexual feeling and its gratification, and to encourage heterosexual feeling. On the other hand, the person of tainted constitution does everything in his power to foster, indulge and cultivate the perverse instinct, while in such a soil the feelings themselves acquire monstrous force. That the future development of this perversity is due to cultivation there is no question. We have only to read the autobiographies to be convinced of it. Thus may arise a perversity that had its origin in a normal reflex, but the accidental cause of which is forgotten with much else of the psychical life of childhood, or, if not forgotten, considered abnormal because of its future monstrous development. Such a reflex, it may be said, if normal, is congenital. This much is in strictness true, but an entirely different aspect is given to the congenital theory. What is really pathological in this aberration is the extraordinary intensification of the sexual feelings and the unbridled lack of restraint with which the subject indulges his senses and seeks every opportunity for gratification. These, without doubt, depend upon the neuropathic constitution. The contrast in this respect with normal heterosexual persons brings the difference into strong relief.

Finally, the fact must not be lost sight of—it is not questioned—that cultivation is capable of generating this aberration and developing it to its most intense degree,

even to the feeling of repulsion for the opposite sex and to the acquisition of contrary tastes and habits. Acquired cases of this kind are recognized and illustrated by cases 94, 95, 96, 99, etc., of von Krafft-Ebing. It is not, then, a question of the sufficiency of this influence. The only question is, "Are all cases due to this influence, or are those cases in which there is no evidence in the histories, *so far as obtained*, of cultivation, and in which there is an apparent spontaneous origin, properly to be regarded as congenital?"

One logical consequence of the cultivation theory has been overlooked, as it seems to me, by von Schrenck-Notzing. It follows as a necessary corollary that this so-called perversion is not really a perversion, but a perversity—a vice rather than a disease.

From one standpoint the view may be modified. It has already been said that a habit may be so intensely cultivated as to become in time almost automatic and independent of volitional control. The nervous processes involved may thus become shunted off from the rest of the psychical life as true psychoses. It is tenable that in some persons these aberrations may become by cultivation real imperative sensations and ideas. Though vice may be the road traversed, the final stage may be disease.

Analogy with what takes place in other fields of the nervous system would make it intelligible that sexual feelings and actions may by constant repetition (cultivation) become associated together and developed into a sort of quasi-independent neural activities, which may then become practically independent of the will—or, in other words, a psychosis.

Sexual perversion, then, may, from the point of view of pathogenesis, be put in the same class with many of the manifestations of hysteria and other psycho-neuropathic states. The constant excitation of various bodily symptoms by the neurasthenic tends to cultivate them into imperative habits, which control his organism. The hysteric dwelling on certain ideas, whether they relate to

herself or her environment, tends to nurture and cultivate them, till they may acquire such monstrous intensification that they control her psychical life.

From small beginnings it is possible that even most intense doubts and fears may be evolved by this cultivation, culminating, perhaps, in imperative ideas (insanity of doubt, folie de toucher, etc.). By constant indulgence of her feelings—revelling in morbid introspection—giving herself up to egotistical debauches, self-pity and wrong inferences, the hysteric or neurasthenic cultivates her body and mind into becoming such a sensitive machine that she can no longer adapt herself to her environment, but must be removed to an institution, where her environment can be adapted to her; of course, I am drawing an extreme picture, but such extreme pictures exist.

Therapeutically, the point of view which we take of the genesis of these psychoses, sexual and non-sexual, is of extreme importance. If they are not the manifestations of a diseased nervous system, in the sense that they are the necessary expression of a diseased body, whether congenital or not, then there is no escape from therapeutic hopelessness so long as the psychopathic state continues. But if psychoses of this kind are the result of cultivation, whether by the influence of external surroundings or by the subject's own conduct—cultivated into psychoses because the soil is a psychopathic one—then we may fairly hope by *counter-education* to replace the morbid processes by healthy ones. For myself, I believe that cultivation is the road by which many neurasthenics and hysterics reach the final goal of confirmed invalidism—cultivated sometimes by the patient herself, too often by the timid physician, who dares not speak the truth, or who fears to do harm. I do not believe in, and I am afraid I have no patience with the *laissez-faire* system which contents itself with removing every source of external irritation and then—doing nothing. The neurasthenic will be more easily

cured if she is isolated, but after this her treatment must be a process of education. The hysteric will do better if her environment is adapted to her weakness, but her education must then begin. I am aware that when the damage is too great nothing will avail—but we can always try. The physician is capable of doing the greatest good—but he is also capable of doing the greatest harm—and no influence for harm is greater than that of the physician who unwittingly encourages corporeal symptoms by intimation of possible disease, danger and caution, or of psychical symptoms by a failure to combat fixed ideas, and to develop a sense of duty, self-control and right-mindedness. A harmful influence of this kind is multiplied a hundred fold by the unremitting kindness and attention and devotion of the physician himself.

Coming now to hard facts, the treatment of the sexual psychoses—if we can accept the statistics of von Schrenck-Notzing—has given results which contradict the congenital theory. Far from being hopeless, as the congenital theory would imply, the treatment of sexual paræsthesia is attended in a large proportion of cases with encouraging results. When the sexual aberration is only a part of great mental degeneration, such as imbecility, dementia or paranoia, of course, any attempt must be hopeless. But where the psychopathic basis is of a minor degree, and the intellect is not materially affected, it must appear, if we are to judge by reports of published cases, that improvement or cure may be accomplished. This, of course, presupposes that a person desires to be cured. It is highly improbable that a person can be cured against his will, and it is evident that many do not want to be cured. The chief and most effective therapeutic remedy has been hypnotic suggestion. In the hands of von Schrenck-Notzing and others this remedy has given decidedly favorable results. The total number of cases collected by von Schrenck-Notzing is 32. The results of treatment were as follows:

Failures.....	5—15.625	per cent.
Slightly improved.....	4—12.5	per cent.
Essentially improved, with later report....	11—34.375	per cent.
Cured, 10; without later report, 2.....	12—37.5	per cent.

100

Thus, about 70 per cent. were essentially improved or cured. The fact that of the 12 cures later reports were obtained, sometimes after considerable periods (four or five years) in 10 makes these statistics of considerable value. Of the 32 patients, 5 were not amenable to hypnosis, 7 were cases of psycho-sexual hermaphroditism, 20 of contrary sexual instinct, 2 of sadism, 3 of masochism.

But, besides direct suggestion, other forms of mental therapeutics should be employed for the purpose of strengthening the will power and developing the character of the patient.

To be brief, the methods of treatment and the results are the same as when dealing with other manifestations of neurasthenia, hysteria or degeneracy, when these are cultivated, as is more than frequently the case. For these, also, isolation is not enough, nor is the so-called rest cure or forced feeding, but education is essential—education in a broad sense—which includes the development of the common sense, the intelligence, the will, the moral sense and the building of the character of the individual.

Dr. William Hirsch submitted a paper entitled:
 NOTES ON A CASE OF TRAUMATIC INJURY OF THE
 PNEUMOGASTRIC, HYPOGLOSSAL, AND SYMPATHETIC
 NERVES,

as a candidate's paper. It is published elsewhere.

Dr. H. M. Thomas submitted a paper entitled:
 PROGRESSIVE CENTRAL MUSCULAR ATROPHY; REPORT
 OF A CASE WITH AUTOPSY,

as a candidate's paper. It is published elsewhere.

DISSOCIATION OF SENSATION OF THE SYRINGOMYELIC TYPE: OCCURRING IN POTT'S DISEASE.

By DAVID LINN EDSALL, M.D.,

Associate of the William Pepper Laboratory of Clinical Medicine; Physician to St. Christopher's Hospital for Children.

I present this report solely because of the peculiar character and distribution of the sensory changes.

The observations which I record were made upon a boy, 14 years of age, whose family history has no relation with the origin of his disease, there being no tuberculosis in the family. The boy's personal history is, excepting an attack of mumps in early childhood, entirely free from disease up to last February, when, while carrying a heavy basket, he felt sudden, sharp pain in the right shoulder and around both sides of the chest, which continued. The diagnosis of pleurisy and also of spinal injury had been made, but the pain was undoubtedly due solely to the spinal caries, as he had no other subjective symptoms of pleurisy, and has now no physical signs of such disease remaining. His pain gradually ceased after resting, and disappeared after six weeks or two months. It has only returned as twinges about either side of the chest, and no pain has been felt for several months. In the early summer he rode a bicycle excessively, but discontinued this in August, as he felt weak in his legs. This weakness was succeeded by stiffness, which increased rapidly, until he walked only with the aid of two canes and partly supported by a companion, moving the right leg with some freedom, but with an extremely spastic gait, while the left leg was very spastic and almost powerless. He was emaciated and emotional. When sitting or lying he could

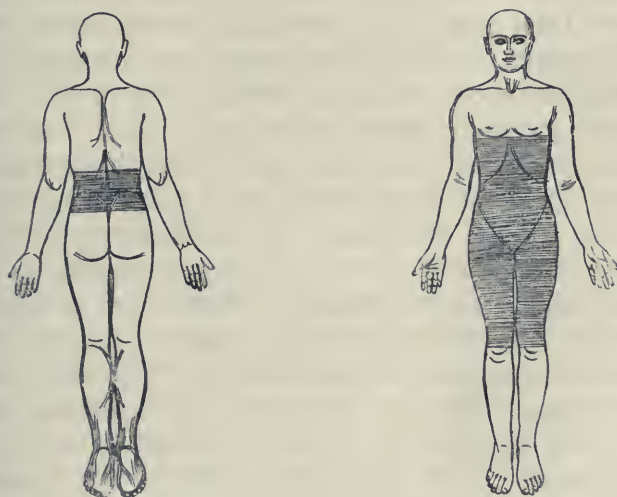
raise the right leg a little and had some power of extension and flexion at the joints. The left leg was almost entirely helpless. The right patellar reflex was violent. On the left it was much excited, but less excessive than on the right. The abdominal, cremasteric and plantar reflexes were absent. Ankle-clonus was present on both sides, and was very forcible. There was no evident atrophy of the muscles. The skin was dry, rough and ill-nourished below the knees. He had then no difficulty with bladder or rectum.

Examination of his spine showed quite pronounced posterior projection of the spines of the second, third and fourth lumbar processes, with some tenderness on pressure upon them.

The sensory condition was the most interesting feature. Tactile sense was entirely preserved everywhere, while pain sense, though still present upon energetic stimulation, was much lessened in a peculiar area, and it required quite severe procedures to cause him pain in this region. Anteriorly, the analgesia began on a horizontal line, which was on a level, in the centre of the body, with the fifth intercostal space, and extended from here down to the level of the knee joint, including all the skin space between these points. Posteriorly, the upper level was at a lower point by about two inches, and extended only one-third the way down the buttocks. Below this the posterior skin surface showed no loss of sensation of any form. The same area that exhibited a lessened pain sense showed an absolute loss of discriminative temperature sense, even to objects which were very hot or almost ice-cold. Whatever the temperature, he inclined to call things "a little warm," though this feeling of slight warmth was not constantly produced.

This examination was confirmed on several occasions by Dr. Willard, who took charge of him in the University Hospital. Just before his admission he had developed some incontinence of urine and of feces. By October 7th,

after he had been entirely at rest, with head extension, for two months, he had regained control of his sphincters, and still retains normal control. Muscular power in his legs seemed rather better, especially in the right leg. The knee-jerks were not noticeably changed, and ankle-clonus was still energetic on either side. Reactions of degeneration were not present. The muscles responded to the faradic current. His sensation was nearly normal, though over the areas that have been described pain sense still seemed lessened, and he had a good deal of hesitancy in



The Area of Sensory Disturbances.

discriminating between hot and cold, and often gave improper answers when the difference in temperature was very marked, and was readily appreciated on any other skin surface than the one indicated.

This improvement did not continue, however, and two weeks later his motor symptoms had increased, and the old dissociation of sensation had returned. By November 15th he was entirely paraplegic; the original condition of the knee-jerks and ankle-clonus persisted, but the hypalgesia and thermoanesthesia had extended down over the

legs and feet and, below the upper limit of the sensory disturbance, the only portion of the surface that showed no change was a posterior area on each side, extending from the upper third of the buttocks down nearly to the knees. At the present time this surface is partly anesthetic to pain and does not discriminate between various temperatures, and he remains entirely paraplegic, though in much improved general health.

The appearance of this form of dissociation of sensation, or the existence of thermo-anesthesia alone in Pott's disease, is of some interest, as very little attention seems to have been given to it. Almost all of the studies of sensory changes in Pott's disease have been confined to the determination of disturbance of the tactile or pain senses. Kocher included several cases of this affection in his studies on spinal anesthesia, and Chipault carefully recorded the areas of anesthesia in 22 cases of spondylitis, but they did not investigate the condition of the thermal sense. Several of the text books on nervous diseases make no allusion to the occurrence of dissociation of sensation in Pott's disease, other than to mention that it may be found in compression myelitis from various causes. Gowers and Strümpell say that all the forms of sensation may be destroyed together, or, more frequently, tactile or pain sense alone. Schlesinger states that he has frequently met with thermo-anesthesia in spinal caries, and refers to Gowers and Bruns as making the same assertion. The individual case which Schlesinger casually mentions and one recorded by Daxenberger are the only cases found by me in detail. In the latter there was loss of pain and temperature sense in the right hand. In this case in the left leg there was at first loss of all sensation; later the temperature sense alone was absent. This difficulty in finding records like my own seems, however, due purely to the lack of investigation into the thermal sense. Isolated disturbance of this sense, or its loss with loss of pain sense, occurs in myelitis as in Biernacki's cases, in spinal tumor,

in tabes, in disseminated sclerosis and in lesions of the nerve roots and of the peripheral nerves, besides being more frequently met with in syringomyelia and hysteria. So that we cannot be surprised at its occurrence in a disease where organic changes are so common as in Pott's disease. It is, however, rather curious that so little attention has been given to it by authors.

More difficult to understand than the occurrence of this dissociation of sensation is its distribution in this case. It does not correspond to spinal segments, nor is it a typical example of the peculiar regional form so commonly found in syringomyelia, and which has been the recent subject of discussion by Chipault and Knapp, since it did not encircle the leg below, but was more extensive over the front than posteriorly. Beyond the fact that it does not correspond to the usual limitations in lesions of spinal segments, as Thorburn, Starr, Kocher and others have determined these limits, it would seem that compression must have acted with a peculiar nicety to have so pressed upon the posterior nerve roots throughout this extent, and in each case have destroyed only pain and temperature sense. So that we are practically forced to accept some change in the cord itself as the cause of this. It accords fairly well with Chipault's division of a combined nerve root and medullary lesion, in which division he adopts the hypothesis of Brissaud that a certain circular level of the cord supplies a corresponding circular area of the limbs or body. This is, of course, purely hypothesis, but is made somewhat more forcible by his discovery in five of the 22 cases he examined that when the caries was not situated over the cord, but over the cauda, he got only the root type. When the caries was situated over the cord itself the medullary type was found alone or combined with the root type, or in certain cases neither type occurred. The root type occurred alone with such situation of the lesion in but two cases, and these two cases showed fair evidence of involvement of the roots alone

without damaging the cord. I have examined quite a large number of records of distribution of anesthesia, and, while that variety which Chipault and Brissaud term the root type may occur alone when the disease is seemingly limited to the cord, I have not, on the other hand, seen any records which showed the medullary type either alone or combined in which there was probably no lesion of the cord. This is not positive evidence, but points to the cord as the probable situation of at least a portion of the disease.

If the cord be diseased in this case—beyond the probable implication of the roots—it is an interesting question whether we have here simply a compression myelitis, an extension of the tuberculous disease into the cord, or a central conglomerate tubercle of the cord.

This is a question which cannot be finally answered in life. Compression myelitis alone might, of course, involve areas of the cord, the disease of which would give rise to the symptoms, as could extension of tubercular disease into the cord. A good explanation of the progress of the sensory disturbance downward would seem to be the existence of some originally central lesion, which gave rise to the early peculiar distribution of hypalgesia and thermo-anesthesia, which lesion subsequently extended outward, so that the fibres conducting temperature and pain sensations from the portions below the area first affected have since been cut off, and a complete loss of these senses has resulted in the corresponding parts. This could be most easily accomplished by a central, isolated tubercle, as either simple compression myelitis or tubercular myelitis could do this only with difficulty and with peculiar progress. Very possibly, if such is the case, the motor symptoms may be largely caused by compression from the nerves and exudate.

Such cases of isolated conglomerate tubercle have been described by Chvostek, Gerhardt, Sachs, Herter, Schlesinger and others, and are well established as occasional,

though rare, occurrences, and have been diagnosed during life.

Beyond the conditions mentioned, the only other possibility of much interest in diagnosis is that of the existence of hysteria with the spondylitis, and that the peculiar distribution of the sensory changes is explained by coincidence. As the fields of vision are of normal extent and the color fields are normal, and all other hysterical stigmata, except this peculiar symptom, are absent, I think the claim that the sensory changes are hysterical would be, as Knapp says of such cases, "begging the question."

REFERENCES.

- Schlesinger: *Deutsche Zeitschr. f. Nervenheilk.*, vol. viii.
Chipault: *Presse Medicale*, Sept. 23d and 30th, 1896.
Brissaud: "*Lec. sur les Mal. Nerv.*," p. 219.
Kocher: *Mittheilungen aus den Grenzgebieten der Medizin u. Chir.*, i., 4.
Starr: *American Jour. of the Med. Sciences*, July, 1892.
Knapp: *Journal of Nerv. and Mental Disease*, Sept., 1897.
Daxenberger: *Deutsche Zeitschr. f. Nervenheilk.*, vol. iv.
Biernacki: *The same*, vol. x., 3 and 4.
Burgess: *Lancet*, Nov. 13th, 1897, p. 1,253.
Chvostek: Abstracted by Schlesinger, loc. cit.
Gerhardt: Abstracted by Schlesinger, loc. cit.
Sachs: Abstracted by Schlesinger, loc. cit.
Herter: Abstracted by Schlesinger, loc. cit.
Gowers: "*Diseases of the Nervous System.*"
Strümpell: "*Spec. Path. u. Ther.*"

Society Reports.

NEW YORK NEUROLOGICAL SOCIETY.

Stated Meeting, March 8th, 1898.

B. Sachs, M. D., President.

Dr. William Hirsch presented a baby of fifteen months, who had been referred to him about one week ago because of certain movements of the head. The child had been perfectly healthy up to about two months ago, when it had fallen from a chair, and struck the back of the head. Vomiting set in immediately afterward, and lasted for about a week. The mother then noticed the peculiar shaking of the head, which has kept up ever since. Examination of the nervous system is absolutely negative. On examining the eyes, one observes that there is a nearly absolute unilateral nystagmus. There is a horizontal nystagmus of the left eye, while the right eye shows the same condition, though very much less marked. The speaker said that it had been claimed by some that these movements in children were brought about by an effort at compensation for the nystagmus. A point in favor of this theory was that the movements cease during sleep. Others had claimed that the movements of the head and the nystagmus had a common cause—e. g., cerebellar lesion. None of the reported cases shows the difference existing in the two eyes as in this case.

Dr. W. M. Leszynsky said that he had seen quite a number of these cases. A great many of them had been found to have ocular defects. In this case, the injury was probably only a coincidence. When the child gets old enough to fix the eyes on objects, these movements are prone to occur. Some years ago he had seen such a case at the Manhattan Eye and Ear Hospital. It was thought that the child was blind and had optic atrophy. He had kept the child under his observation for seven or eight months. It improved under tonic treatment, and ultimately recovered

completely. He did not believe, therefore, that there was any actual morbid change in these cases.

Dr. George W. Jacoby said that he had not found these cases uncommon, and he believed that they occur directly as a consequence of traumatism. There were two distinct classes of cases, i. e., those with nystagmus, and those without it. In the majority of cases, by excluding vision, it would be found that the movements of the eye cease. In this child there seemed to him to be some up and down movement interspersed with the other movements. Some years ago he had seen an interesting medico-legal case, occurring in an adult—a man who had been struck in the eye by an electric light wire. The eye had been perforated. After losing the eyeball he presented certain psychic symptoms, together with some head nodding, which he had continued ever since. He was now rather feeble-minded. Inasmuch as in this man there was nothing pointing to injury of any other part of the brain except the frontal lobe, the question arose as to whether there was any possible connection between the injury to this lobe and these movements.

Dr. Frederick Peterson said he believed Henoch first described these movements, many years ago. He had himself described ten cases, some years ago, as gyrospasms, representing all the different varieties. Most of the cases have a lateral movement, but some have the head nodding. Henoch thought the condition was a reflex from dentition, as the majority of the cases developed at about the age of eight or ten months. The theory that he had adopted was that these cases were almost always the result of a trauma or concussion. A point worthy of note was that there could not be any marked pathological lesion, as all of them recovered. The lateral movement of the head and the movement of the eyes seemed to him to be due to functional troubles in different parts of the brain. Many of these cases would temporarily stop the movement if the eyes were fixed on an object. If the lateral movement were due to nystagmus, it was curious that the nystagmus should be present in the head-nodding movement.

Dr. Mary Putnam-Jacobi said that many years ago she had seen a case coming on spontaneously in a child of two years. It was peculiar in that a rotary movement came on every night at bedtime.

Dr. Fraenkel said that he had recently seen a child of nine months, who, according to the mother, had exhibited a rotary movement of the head and a vertical nystagmus since it was three months old. This nystagmus occurred when the nodding movements ceased. This would seem to support the view of Dr. Leszynsky, that it is dependent upon some refractive condition of the eye.

Dr. Hirsch said that the objective examination by Dr. Koller showed the eyes to be entirely normal. All the theories offered would not hold good in a case of unilateral nystagmus. That the condition in the present instance was caused by traumatism alone seemed almost certain from the history. The movements of the head are entirely separate from those of the eyeball. The fact that most of the children recover was sufficient to exclude an organic lesion, but a slight pachymeningitis in the region of the cerebellum might recover.

Dr. Fraenkel presented a child, two and a half years old, born of healthy parents. The family history is negative. The child was born at full term, by the breech, after a rather difficult labor. Immediately after its birth the abnormal condition of the lower extremities was noted. The mother insists that the child has improved. Examination shows a moderate lateral curvature, and the child is unable to sit, walk or stand. Electrical examination shows extensive degeneration of all the muscles except the calves. The knee-jerks are absent, but there is an ankle clonus on both sides. At first sight, the case seemed to be one of poliomyelitis, but it was conceivable that the condition was the result of a dropsy, or of a hemorrhage from rupture of the anterior spinal artery. As this vessel supplies the anterior horn almost exclusively, it was possible that a traumatic poliomyelitis had developed, extending from the mid-dorsal to about the third lumbar segment. Or, there might be a localized cavity formation. He would explain the existence of the ankle clonus by the theory that, as the calf muscles were in a state of increased tonus, due to the fact that the antagonists were gone, the moment a sensory stimulus was applied, and entered the cord, the latter being in a state of increased receptivity; the evidence of its having received this stimulus was shown by an additional motor discharge. The spinal curvature was to be explained by the unilateral involvement of the spinal muscles. The posterior muscles below the knee gave an exaggerated response with both electric currents, while there was no reaction in the muscles above the knee.

Dr. Peterson asked why this explanation of the ankle clonus would not apply to all other cases of poliomyelitis, in which, it was well known, there was no ankle clonus.

Dr. Fraenkel replied that the presence or absence of ankle clonus was dependent upon quantitative changes in the tonus

of the muscles. The increased tonus of the calf muscles only he did not think existed ordinarily in cases of poliomyelitis.

Dr. Terriberry said that the marked extension of the foot was seen quite frequently in ordinary poliomyelitis, owing to one set of muscles being left without any antagonizing ones. But the other features of this case were certainly unusual.

Dr. Joseph Collins accepted the pathological explanation given by Dr. Fraenkel. The fact that ankle clonus does not occur in ordinary cases of anterior poliomyelitis did not negative the explanation given. The case was, in reality, a unique substantiation of the theory proposed by Dr. Fraenkel last winter, and adopted by Hughlings Jackson in his lecture this year. In no case of complete anterior poliomyelitis that he had seen had there been any hypertonus of the calf muscles.

Dr. B. Sachs thought there was at least one other view that could be put forward. He did not think that there was this amount of spasticity in a very large number of cases of ordinary poliomyelitis. He had not seen a single instance of ankle clonus in an ordinary case of poliomyelitis; the unusual spastic condition in the present case must be due to some special lesion. It was fair to assume that in this case there was some developmental defect. The entire appearance of the leg reminded him very much of cases in which children had been born with defective limbs. On the supposition that the spinal cord was defective, chiefly in the gray matter in the region of the lumbar segments and in the lateral columns, one could understand how the knee-jerks might be absent and the ankle clonus present.

Dr. Fraenkel said that in estimating the comparative frequency of ankle clonus and of absence of the knee-jerks in ordinary cases of poliomyelitis, it should be remembered that it was exceptional to examine such cases on these points.

Dr. Joseph Collins presented a boy of thirteen years with lateral curvature of the spine, atrophy of the muscles of the right upper extremity and the condition of the face known as the Schultze eye. The only history was that he had begun to have diarrhoea three or four years ago, and that this had persisted pretty constantly for a year or two. He looked upon the case as one of syringomyelia affecting the anterior horns, and he based this diagnosis chiefly on the ocular symptoms on one side, the atrophy of the hand and the spinal curvature. The atrophy of the hand had begun about two years ago.

Dr. G. W. Jacoby said that to base the diagnosis on these three symptoms alone required considerable assurance. In his opinion, such a diagnosis was not warranted, unless there were marked sensory disturbance with the atrophy.

Dr. William Hirsch said that he had recently shown two cases of syringomyelia in another society, which showed the same condition of the muscles and of the eye—the narrowing of the fissure of the eyelid, and the contraction of one pupil. There were also present the typical sensory symptoms, but the atrophy of the ulnar group and the condition of the eye had led him to believe that it was a typical form of syringomyelia located between the last cervical and first dorsal segment. He had also recently seen in private practice a lady with the same condition of the eye, and with a herpes zoster in the trigeminal region, and who also showed instead of an atrophy a tremor of the left arm and a slight analgesia. The diagnosis made by Dr. Collins seemed to him perfectly justified. The other more characteristic symptoms would probably develop later.

Dr. L. Stieglitz agreed with the diagnosis of syringomyelia, and remarked that at the last meeting he had presented a counterpart of this case, in which no sensory symptoms were present, and also a typical case of syringomyelia. He thought it was not uncommon to find these cases without sensory symptoms.

Dr. Terriberry remarked that an injury of an anterior root might produce the atrophy and the ocular symptoms.

Dr. Collins asked at what level this must be to cause sympathetic involvement of the right side of the face with destruction of the sixth, seventh and eighth cervical segments.

Dr. Terriberry replied that he did not think there was any reason why the cord need be considered to be involved.

Dr. Onuf said that in a joint investigation, made by Dr. Collins and himself, they had found that the zone situated between the central ganglia on one side and the end of the lateral horn on the other, and between the base of the posterior and anterior horns, was intimately connected with sympathetic functions. They had found that atrophy of the cells of the lateral horn took place in a certain group (the lateral, the central ganglia, and in the whole zone in between), and that this atrophy was partly in the sensory and partly in motor fibres. The motor fibres originate from the smaller cells of the zone mentioned. This being the case, it was evident that syringomyelia could present very different pictures depending upon the particular locality affected. In the case under discussion the sympathetic symptoms were very marked in connection with the eye, and the diarrhoea and the lateral curvature might also be considered as belonging to the same class. In three cases they had extirpated the stellate ganglion in cats. This was usually followed after some weeks by a diarrhoea, which was most persistent and exhausting. This ganglion is supplied chiefly by the upper dorsal nerves.

Dr. M. Allen Starr said that the combination of the sympathetic paralysis with ulnar nerve paralysis he had known to occur in one case of undoubted gumma on the anterior surface of the cord. The gumma was absorbed after a time. The condition had been undoubtedly produced by the pressure on the anterior nerve roots.

Dr. Pearce Bailey said, regarding the first dorsal root, and its relation to the sympathetic, that over ten years ago Klumpke had made her experiments on animals. She had found that the nutritive fibres for the eyeball were affected by the injury of the first dorsal root, but that there were no accompanying vascular disturbances. This had been substantiated by other observers. The vascular supply of the face goes along the spinal canal externally, and is joined by the first dorsal root at its exit.

Dr. Collins said that he had been largely led to make the diagnosis of syringomyelia in this case by the experiments that he had conducted in conjunction with Dr. Onuf. There was nothing else which would produce the four prominent symptoms, viz., the diarrhoea, curvature of the spine, atrophy of the hand muscles and the Schultze eye.

Dr. Hirsch presented a young woman who was an example of total unilateral congenital sweating of the face. She complained that half of the face would become red and moist, while the left half remained dry and of normal color. She never sweats on the left side of the face. He had experimented with hypodermic injections of different drugs. Physostigmin had had no effect at all. Pilocarpin, injected hypodermically, caused perspiration all over her face, for the first time in her life; but, of course, this was only transient. The fact that in this case absence of perspiration is associated with absence of vasomotor symptoms seemed to be in favor of the view that the vasomotor centres and the sweat centres are at least intimately connected. The condition is confined to the face.

Dr. Starr remarked that a case of this kind, occurring in a man of twenty-three years, had been entirely cured in his clinic by boring out his turbinated bones.

Dr. Fraenkel presented a young boy, who had come under his observation about three months ago with a diagnosis of pulmonary tuberculosis, because of an attack of hæmoptysis. He had been well up to two years ago; then he had begun to suffer from shortness of breath. On admission to the Montefiore Home the head was turned

to one side, there was inspiratory stridor, a moderate amount of exophthalmos, swelling of the neck and tachycardia. The physical examination of the chest was practically negative. The tumefaction in the neck was lobulated, and moved up and down during deglutition. Under treatment with thyroid extract he had decidedly improved subjectively, and had gained fourteen pounds in body weight. The tachycardia had disappeared. The case was interesting as showing the difference between genuine Basedow's disease and the secondary or symptomatic form.

Dr. C. E. Nammack asked if Hodgkin's disease had been excluded.

Dr. Fraenkel replied that a brother of this patient had twice had tuberculous glands removed, and there had been good reason to believe that this might be a case of Hodgkin's disease, but it had been excluded (1) by the absence of other evidence of lymphatic involvement; (2) by its long duration; (3) by examination of the blood, and (4) by the mobility of the swelling in the neck on deglutition, showing its connection with the thyroid gland.

Dr. B. Sachs presented a man, fifty-one years of age, whom he had first seen about one week ago. He had been married twenty-seven years. Six years ago his wife had been afflicted with the same affection as the one he now suffers from. The patient himself had been in good health in former years, and he still weighs 230 pounds. He has been an extremely heavy drinker, chiefly of beer, taking, at times, as much as fifty or sixty glasses a day. Syphilitic infection could not be established. He had been in good health up to January 12th, 1898, when, while attending the funeral of a friend, he says he saw a flash of light, and this was immediately followed by double vision and intense photophobia. At first glance there would appear to be a double ptosis, but the eyelids can be moved upward. Ordinarily they droop in an effort to protect the eyes. There is slight nystagmus and a decided paresis of the left rectus externus. The pupils are irregular; they do not react to light, and but slightly to accommodation. The case had been referred to him by Dr. Marple, who had found nothing, on ocular examination, to explain the photophobia. Further examination showed a very widespread and marked hemi-analgesia, but no impairment of tactile sensibility. He had arrived at the conclusion that there was a large hysterical element in the

case. The visual fields are normal; the reflexes are normal, and there is no evidence of loss of power in the extremities. His diagnosis was hysterical ophthalmoplegia.

Dr. J. Arthur Booth referred to a girl of twenty years, who had had double ptosis, but no ocular paresis, who had been cured by hypnosis.

Dr. Fraenkel said that in a recent monograph hysterical ophthalmoplegias of this character had been described. Aside from the clinical development of the case, its development after emotional disturbance was particularly significant. About six months ago a man, in a very similar condition, had applied for admission to the Home. He presented ataxia, loss of knee-jerks, ptosis and ophthalmoplegia. After admission his ptosis and ophthalmoplegia disappeared, and the case proved to be clearly one of locomotor ataxia, the other symptoms having been hysterical, and added to the others with a view to securing his admission.

Dr. Leszynsky said it was important to distinguish between ptosis and blepharospasm. In the case under discussion there seemed to be a certain amount of blepharospasm. With photophobia tonic blepharospasm was much more likely to occur than ptosis. He had seen a number of such cases in hysterical individuals, and quite recently one in a young girl who responded promptly to hypnosis.

Dr. Peterson referred to a case of bilateral ptosis, or, rather, of tonic blepharospasm, which had presented very much the picture shown in this patient. A cure had been effected by two or three applications of the faradic current.

Dr. Sachs said he proposed to treat this case by suggestion.

Dr. Sachs then presented another case of ophthalmoplegia, in a boy of seventeen years. In October, 1894, at 9 A. M., he had found himself unable to utter words. This had passed away, but had been repeated at noon and at 4 P. M. He had then a convulsion lasting ten minutes, after which the left eyelid had been noticed to droop. There had been no convulsions since then. He had been perfectly well previously. Examination showed ptosis of the left eye and slight ptosis of the right eye. The outward and inward movements were limited in single and conjugate action; both pupils reacted well to light and accommodation; the sensation of the face was normal. There had been comparatively little change in the past three years. There is diplopia, chiefly when looking to the left. The diagnosis lay between thrombosis or embolism in the basilar artery. The heart action is irregular and rather rapid, but no murmur is audible. Six years ago

he was under treatment for a time for cardiac palpitation.

Dr. Sachs also presented a man, thirty-nine years of age, who had been admitted to the Montefiore Home some time ago. There was no evidence of syphilis. At the age of twenty he was weak in the knees and frequently made missteps. In 1887 he had sought medical advice because of difficulty of locomotion, noticed especially in climbing stairs. When examined in March, 1895, he complained chiefly of difficulty in walking, weakness in the extremities and slight difficulty in speech. At first the case was supposed to be one of locomotor ataxia. He now has an ataxic spastic gait, and also has static ataxia; the pupils react to light and during accommodation; the patellar reflexes are absent. There is no Argyll-Robertson pupil. There is distinct ataxia of the right upper extremity. He has a form of speech which is midway between a slow speech and a bulbar speech. The diagnosis lies between a bulbar form of multiple sclerosis and the possibility of Friedreich's ataxia instead of an ordinary tabes. The great point against Friedreich's ataxia was its occurrence rather late in life; while the absence of the knee-jerks militated against multiple sclerosis. There was no disturbance of sensation. The jaw-jerk is absent.

Dr. Fraenkel said that, although the lack of co-ordination was first noticed at the age of nineteen, when his attention was naturally directed to it by entering the army, it was not improbable that it had been present long before. There was also slight atrophy of the optic nerves. The absence of sexual and sphincteric disturbance and the peculiar thick and scanning speech seemed to point rather to the diagnosis of multiple sclerosis.

Dr. Collins said that when he had first seen the man, three years ago, the intention tremor and the optic atrophy had not been present, and it was then thought that the man had Friedreich's disease. In the last two years the speech had become very much more bulbar in quality. He was inclined to believe that there was a diffuse insular sclerosis, bulbar and spinal.

Dr. Hirsch said that even at the present time there was not a perfect agreement as to what constitutes the pathological basis of Friedreich's disease. The case seemed to him like the cerebellar form of Friedreich's disease.

Dr. Sachs said that the term "Friedreich's disease" is at the present time applied usually to the ordinary hereditary ataxia, the disease being located partly in the posterior, and partly in the lateral columns of the cord.

PHILADELPHIA NEUROLOGICAL SOCIETY.

January 24th, 1898.

The President, Dr. Charles W. Burr, in the chair.

Dr. F. X. Dercum exhibited

A CASE OF HEMIALGIA.

The patient was a well-developed man of thirty-seven years, a native of Poland and a laborer by occupation. He had been in this country seven years, and had always worked in iron works. During this period he had been exposed to very high temperatures. His family history was negative. He had had none of the diseases of childhood, and had always been in good health until a little more than a year ago, when the present trouble began. He denied venereal disease and alcoholism. He first noticed pain in his right knee, which, after six months, became so severe that he was obliged to give up work. Shortly afterward the whole of the right thigh began to ache, and later on this aching involved the right half of the trunk, especially the chest, the right upper limb, and the right side of the face. The knee presented no objective features worthy of note. The physical examination of the chest revealed nothing abnormal. The knee-jerks on both sides were much exaggerated. The right pupil was slightly larger than the left, but the eyes reacted normally to light. Later it was noted that the patient had less muscular force in the arm and leg of the right side than in those of the left.

Dr. Tyson, who had had charge of the patient, being unable to arrive at a definite conclusion with regard to the case, termed it one of hemialgia, the pain being the most conspicuous symptom presented.

After the patient had been in the hospital five months, he was transferred to the service of Dr. Dercum. Examination now showed that Romberg's sign was absent. The man stood readily upon the left leg alone, but was

unable to stand alone upon the right. The gait revealed decided weakness of the right leg, which was especially marked when the patient was asked to walk backward. Both knee-jerks were exaggerated, but especially the right, and a distinct ankle clonus was also elicited upon the right side. There was no ataxia of the legs or hands. There was no loss of the tactile sense or the thermal sense, and no analgesia. Fibrillary tremors were noticed in the quadriceps extensor muscles of both sides, and in the gastrocnemius of the right leg only, but they were not present in the arms. There were also signs of marked vasomotor relaxation. The thighs, legs and feet of the patient were mottled and livid, and pin pricks produced bright pinkish areas. The arms showed, to a slight degree, the mottled and flushed condition seen in the legs. This was slightly more pronounced in the right arm, and rather more noticeable in the forearms and hands. The muscles and tendon reflexes in the arms were also exaggerated upon the right side.

The trunk anteriorly and posteriorly presented a similar condition of vasomotor relaxation. Marked tache cérébrale was also noted in the back. The patient wore a flannel binder and gave as a reason the pain and stiffness in the back. Girdle pains were absent. Exaggerated irritability was also observed in both pectoral muscles, and in both scapular groups. The tongue was protruded in the median line but with slight tremor. There was slight tremor of the lips upon pouting. At this examination the pupils were equal and responsive to light. An ophthalmic examination by Dr. de Schweinitz was negative. The skin reflexes revealed no change.

At present, Jan. 24th, 1898, the patient still complains of severe pains in the entire right half of the body, but the signs of motor weakness, quite distinct and notable some months ago, have now almost entirely disappeared. Peripheral causes of pain can readily be excluded. No pain is caused by pressure on the nerve trunks or by movement. There are no signs of hysteria. The symptoms suggest an organic hemiplegia with a lesion in such a situation as to give rise to the symptom of pain. Dr. Dercum stated that his case was unique in his experience, because the hemiplegia was relatively slight while the pain was excessive. He thought it hardly safe to speculate regarding the nature of the lesion. Headache and other symp-

toms of brain tumor were absent, and for the present he could but coincide with Dr. Tyson in terming this case, in accordance with the principal symptom present, one of hemialgia.

Dr. Charles K. Mills said that this case was interesting in connection with the recorded cases of pain associated with hemiplegia, which, as is well known, are of at least two classes. In one set of cases the pain is undoubtedly due to peripheral conditions like neuritis. Again, as has been recorded by Weir Mitchell and others, we have cases in which, preceding, accompanying or following the hemiplegic attack, pain on the paralyzed side is a marked feature. He had seen a few cases of this description in which there were no evidences of local conditions to account for the pain. He referred to a case reported by Edinger, in which pain was found to have been due to a cerebral lesion, and he thought that Dr. Dercum's case might be one in which the hemialgia was caused by an irritative lesion in some portion of the cerebrum, possibly in the cerebral sensory pathway, or in the thalamus.

Dr. A. A. Eshner spoke of the possibility of there having been some acute infective or inflammatory process at the beginning of the trouble. The distribution of the symptoms, the presence of pain, the primary loss of motility, and the increase of the knee-jerk, suggested cerebro-spinal meningitis to him. He acknowledged that this diagnosis might seem far-fetched, but it would explain the symptoms, as well as any other, and would not be in discord with the ocular manifestations.

Dr. Wm. Pepper, Jr., presented for Dr. Mills

THE BRAIN FROM A CASE OF BILATERAL SYMMETRICAL
SOFTENING OF THE INTERNAL CAPSULE,

AND

THE BRAIN FROM A CASE OF CEREBELLAR TUMOR.

The first patient had had bilateral hemiplegia, and the second had complained of occipital headache, staggering gait and stupor. At the necropsy of the second case several masses, probably tubercles, were found in the right lobe of the cerebellum.

Dr. Charles K. Mills said that instances of bilateral softening are rare. There were three apoplectic attacks in this first case; one seven or eight years ago, a second two or three years later, and a third ten or twelve days ago.

The second patient was supposed to have been syphilitic, and probably the diagnosis was correct, but he was also the victim of wide-spread tuberculosis. He had been salivated without the slightest improvement. Dr. Mills spoke of the importance of bearing in mind the possibility of tuberculosis in cases of this kind.

Dr. Wm. Pepper, Jr., exhibited for Dr. Dercum

THE BRAIN FROM A CASE OF CEREBRAL ABSCESS.

Convulsions, progressive weakness of the right side of the body, stupor, some hyperpyrexia, though usually a normal temperature, and negative ophthalmoscopic findings, had been noted in this case. Two communicating abscesses were found in the upper part of the left parietal lobe at the necropsy.

Dr. Dercum said that abscesses, especially large ones, are infrequent in this situation, and that when they do occur they are generally multiple. In his case there was one large abscess, together with another smaller one. Had abscess in this case been diagnosticated, it is probable that the larger one could have been successfully treated by surgical means; but even then it is extremely probable that the smaller one would not have been found. He had been led astray by the history of specific infection. The rise of temperature was regarded as due to purulent cystitis and pyelitis. The subnormal temperature, so frequently present in abscess of the brain, was not present here. Just what the source of infection was, it is difficult to say. There was no lesion in the lungs or pleural cavity; no endocarditis, and there were none of the ordinary sources of infection which give rise to cerebral abscess. We must remember, however, that not infrequently cerebral abscess follows a wound of the external tegument, and may not produce symptoms until long after the wound has healed and been forgotten. This woman had been operated upon for pelvic disease, which was probably some purulent affection.

Dr. Dercum recalled the case of a young man who was admitted to the University Hospital with a stab wound of the left side. The wound healed, and months afterward the man was readmitted to the hospital, presenting symptoms of organic cerebral disease. In this instance, as in the previous one, cerebral syphilis was diagnosticated, the diagnosis being based largely upon the obscure character of the symptoms and the history of the specific infection. At the autopsy a

large abscess, involving one hemisphere, was discovered. No source of infection could be detected, but Dr. Dercum could not dissociate the stab wound of the side and the affection from which the man died.

Dr. Spiller said that the temporal lobe is the most common location of cerebral abscess, and that Körner, in a critical digest on the recent literature of this subject, had stated that Oppenheim, Pick and Manasse had observed optic aphasia in abscess of this portion of the brain. It seems to be caused by the location of the abscess in the posterior part of the second and third temporal gyri.

Oppenheim, in his *Lehrbuch*, says that pure cases of optic aphasia have not been studied post-mortem, but they seem to be due to lesions at the junction of the left occipital lobe with the temporal, when they are sufficiently extensive to interrupt the fibres passing from both occipital lobes to the left first temporal convolution. Oppenheim observed three cases of optic aphasia. In the first, an instrument penetrated the skull during an operation on the ear, and probably injured the anterior part of the occipital lobe. The optic aphasia was only transitory. In the second case, optic aphasia, with right hemianopsia, was the first sign of a tumor in the left parietal lobe, which almost entirely separated the temporal from the occipital lobe. In the third case, a tumor was situated in the basal part of the occipito-temporal convolutions.

Dr. Spiller said that the case reported some time ago by Dr. Mills and Dr. McConnell, as a proof of the existence of a "naming centre," was one of optic aphasia associated with tactile aphasia, and that in this the lesion—a tumor—had also been found in the lower posterior part of the left temporal lobe. We have, therefore, considerable evidence regarding the location of a lesion producing optic aphasia.

Dr. J. W. McConnell reported

A CASE OF NEURITIS OF THE FIFTH NERVE WITH
HERPES AND ECZEMA.

The patient was a white female, aged about sixty years, married, a native of America, and had always been in good health until the present illness. A shampooing of the scalp and exposure to night air were followed by pain in the distribution of the first division of the left fifth nerve, and later by a herpetic eruption over the area of the pain. There was no pain elsewhere in the body, except in the preauricular and cervical glands of the left side, which

were enlarged and tender. A few days later an eruption of fine vesicles appeared over the skin of the affected area, which was previously healthy. These were moist, inclined to crust, and attended with burning and itching. Anæsthesia of the diseased locality was found, but there was no motor paralysis. The patient at this time complained of general pains, and a slight rise in temperature was demonstrable. The acute symptoms lasted three weeks, leaving, on their disappearance, discoloration of the skin, anæsthesia, dull pain and soreness; the latter continuing for nearly a year.

Several interesting features were presented by the case. The patient had years before suffered an attack of "shingles," recovery from which, however, was good. The writer could not find any account of an eruption of eczema occurring simultaneously with an attack of herpes. He considered the limitation of the eczematous eruption to the distribution of one nerve a rather unusual circumstance. The occurrence of adenopathies, either local or general, he argued, might support, in a degree, the theory of the infectious nature or origin of herpes; and, in view of the prevalence of influenza at the time of the patient's illness, and the fact that she presented some symptoms of that disease, he was led to believe that the skin conditions were dependent upon a neuritis, which probably had as its exciting cause an attack of influenza.

Dr. J. H. W. Rhein reported

A CASE OF UNILATERAL SWEATING AND FLUSHING
OF THE FACE.

The patient had periodic attacks of pain in the right arm and hand, with perspiration and flushing of the right side of the face.

Dr. Charles W. Burr reported

A CASE OF DISTURBANCE OF GAIT DUE TO A DELUSION.

This man would take a few short steps and be unable to advance further until after waiting a minute or two,

or after some words of encouragement, he was able to make another attempt. He said that he feared that there might be an opening in the floor into which he would fall. Dr. Burr thought that the abasia was due to the man's mental condition.

Dr. Burr also presented

A CASE OF ASTASIA-ABASIA.

This woman was able to walk when in the open air, but could not do so when she was within the house. He regarded the case as one of hysteria.

Dr. Dercum said he had repeatedly examined the first case described by Dr. Burr. He believed it to be due to a mental condition. The man seemed to be very much afraid. When urged, he would walk a short distance, stop, and then tremble with fear. The gait he presented suggested similar gaits which we sometimes observe in the wards of insane asylums.

Dr. A. F. Packard and Dr. Alfred Hand, Jr., showed

A SPECIMEN OF MULTIPLE TUMORS OF THE BRAIN

from a mulatto child, aged three and a half years, who had died of tuberculous peritonitis. During life there was absolute paralysis with rigid contracture of the left arm and leg. The palsy and contracture followed immediately after a severe convulsion, which had involved the left face, arm and leg, two and a half months before the death of the child; no change was ever detected in the eye-grounds.

At the autopsy, in addition to the tuberculous peritonitis, three brain tumors were found; one in the posterior parietal region of the right cerebellum, a second in the corresponding portion of the left cerebellum, and a third in the basal ganglia of the right side of the brain, which it had replaced or destroyed.

Dr. Alfred Hand, Jr., said that the intense round-cell infiltration suggested either gumma or tubercle. The absence of the tubercle bacilli would be merely negative evidence. The absence of endarteritis would point more toward the exclusion of gumma than the absence of tubercle bacilli would to the exclusion of tuberculosis.

He had been asked as to the method of preservation of the specimens, and stated that it was the one elaborated by Kaiserling, for which three solutions are necessary.

Solution No. 1 consists of formalin 250 parts, potassium acetate 30 parts, potassium nitrate 10 parts, water 1,000 parts.

Solution No. 2 is alcohol, 85 per cent.

Solution No. 3 is made of potassium acetate 100 parts, glycerine 200 parts, water 1,000 parts.

The specimen is left in No. 1 for from one to five days; in No. 2 until the color returns, in from one to six hours, and then preserved finally in No. 3.

Dr. Spiller spoke of the great value of formalin in the preservation of nervous tissue. Müller's fluid is not thoroughly reliable, especially in warm weather, and when the brain is not cut into pieces; it also stains the tissues, and renders gross lesions much less distinct; it prevents the employment of Nissl's stain, which often is a most serious objection. Formalin (ten parts to ninety parts water), on the other hand, hardens much more quickly, preserves the tissues better, does not stain the material, and, most important of all, permits the use of Nissl's method. If the sections are placed for twenty-four to forty-eight hours in Müller's fluid, they may be stained as well by Weigert's hæmatoxylin method as if they had been originally hardened in the bichromate solution. Every student of the pathology of the nervous system knows the great need of staining sections from the same region, so as to show the cells as well as the fibres.

Dr. Joseph Sailer reported

A CASE OF SECONDARY SUPPURATION IN THE SELLA
TURCICA IN TYPHOID FEVER.

Exophthalmos, among other symptoms, had been noted. At the necropsy purulent phlebitis of the right Sylvian vein, of both cavernous sinuses, and of the left anterior cerebellar vein, as well as suppuration within the sella turcica and retrobulbar abscesses, were found.

Periscope.

With the Assistance of the Following Collaborators:

CHAS. LEWIS ALLEN, M.D., Wash., D.C. R. K. MACALESTER, M.D., N.Y.
 J. S. CHRISTISON, M.D., Chicago, Ill. J. K. MITCHELL, M.D., Phila., Pa.
 A. FREEMAN, M.D., New York. H. PATRICK, M.D., Chicago, Ill.
 S. E. JELLIFFE, M.D., New York. JOSEPH SAILER, M.D., Phila., Pa.
 WM. C. KRAUSS, M.D., Buffalo, N.Y. HENRY L. SHIVELY, M.D., N.Y.
 W. M. LESZYNSKY, M.D., New York A. STERNE, M.D., Indianapolis.

ANATOMY.

78. EINE GOLDFÄRBUNG DES NERVENMARKES. (Staining the Axis Cylinders of Nerves by Gold). M. v. Frey (Archiv f. Anatomie u. Entwicklungsgeschichte, 1897, Supplementband, Dec., p. 108).

The author describes a method which has proven of service to him in the tracing of nerve fibres in the skin, mucous membrane and viscera.

Small pieces of tissue are allowed to lie for two weeks in a 2 per cent. aqueous solution of ammonium bichromate, at a temperature of from 1—5 deg. C. They are then carefully washed in running water for ten or fifteen minutes and placed in a 1 per cent. solution of chloride of gold to which 1 per cent. hydrochloric acid has been added. The length of time required in this solution is determined in large part by individual experiment. The specimens are then washed slightly and the reduction of the gold effected by a one-fiftieth per cent. aqueous solution of chromic acid. Specimens can be cut after twenty-four hours of reduction. The superfluous gold may be removed by sodium hyposulphite solution.

The nerve fibres are stained a bluish green to a bluish black if properly impregnated. JELLIFFE.

79. ZUR KENNTNISS DER PERIPHEREN GEHÖRLEITUNG (On the Peripheral Acoustic Conducting Paths). H. Held (Archiv f. Anatomie u. Entwicklungsgeschichte, 10, 1897, p. 350).

The author holds that the fibres of the ring plexus of the spiral ganglia are collaterals of the peripheral branches of the cochlearis cells; they end in the hair cells of Corti's organ, and again give off from each, collaterals which run forward to other hair cells. Thus hair cells from different parts of Corti's organ are brought into communication with one ganglion cell.

80. DIE NERVENENDIGUNGEN AN DEN TASTHAAREN VON SÄUGETIEREN (The Nerve Endings of the Tactile Hairs in Mammals). E. Botezat (Archiv f. mik. Anat., 50, 1897, p. 142).

The nervous fibres going to the taste follicles are described by the author as being disposed in two sets—a deep and a superficial. The first forms a complicated plexus, by frequent and irregular anastomoses, of varicose fibres pursuing an undulating path and surround-

ing the base of the root sheath, and the basal thickening of the papillæ. The superficial layer consists of ascending longitudinal fibres, which cover the preceding. The fine axis cylinders which come from these two series of fibres traverse the vitreous membrane, and form thick plaques about the tactile cells of Merkel. These plaques are somewhat meniscus shaped; they are not the true nerve terminations, however; these are found as very minute and delicate axis cylinder prolongations, which end between the cells. VOGEL.

81. OBSERVATIONS ON SENSORY NERVE-ENDINGS IN VOLUNTARY MUSCLES. A. Ruffini (Brain, 20, 1897, p. 368).

A series of notes is here presented dealing with muscle spindles, tendon organs and Pacinian corpuscles found in muscles. The general conclusions to be derived from the author's paper are:

1. There are in the voluntary muscles nerve fibres of three kinds—motor, sensory and vaso-motor.

2. The motor nerve fibres end, as is well known, in the *end plates* of Rouget and Kühne.

3. The sensorial nerve fibres possess three quite distinct end organs in man and in all the higher vertebrata. These sensorial end organs of muscle are: (a) The muscle spindles, (b) the tendon organs (or Golgi organs) and (c) Pacinian corpuscles.

4. The vaso-motorial nerve fibres form reticular plexuses or true terminal plates (Mazzoni), or terminate simply on the capillary walls with a fine apical enlargement (Ruffini).

5. The functions of the motor plates and vaso-motorial endings has been known for years. Further, physiological experimentation is now wanted to investigate the functional activities of the three sensorial organs, the spindles, the tendon organs, and the Pacinian corpuscles. In the author's opinion it is to these three kinds of sense organs that physiology must turn its attention if it will resolve the problem of the muscular sense. JELLIFFE.

PATHOLOGY.

82. THE MUSCLE SPINDLE UNDER PATHOLOGICAL CONDITIONS. Fred E. Batten (Brain, 20, 1897, p. 138).

The author treats of the following subjects in this paper: The history of the researches made upon the muscle spindle, the various views held with regard to its origin and function, the technical methods employed in the research, the histology of the spindle as found in man, and its modifications in the following diseases: Infantile paralysis, tabes, myopathy, progressive muscular atrophy, peripheral neuritis, trauma of brachial plexus and following sciatic nerve section.

In these various diseases, *seriatim*, the author states:

In infantile paralysis it would seem probable that the muscle spindle remains absolutely intact, both in regard to the intra-fusal muscle fibres and in regard to the contained nerves.

In tabes, in two cases of three examined, the spindles were normal; in a third there was a mild grade of degeneration.

In myopathy (Leyden form) the spindles seemed increased in number, but there were no traces of degeneration, or other change.

In progressive muscular atrophy the author's observations are in accord with those of other writers who find no changes in the muscle spindle.

In peripheral neuritis, in one case only examined, there were no changes in the muscle spindles.

In injury to the brachial plexus, with loss of motion and sensation of a year's standing there was an atrophy of the muscle spindle.

In experimental sciatic sections in cats atrophy of the spindles was found.

The paper is richly illustrated and a careful bibliography is appended. JELLIFFE.

83. LE PHÉNOMÈNE DE CHROMATOLYSE, CONSECUTIF A LA LESION PATHOLOGIQUE OU EXPERIMENTALE DE L'AXONE (The Phenomena of Chromatolysis, etc.) M. Van Gehuchten (Bulletin de l'Academie Royale de Medicine de Belgique 11, 1897, p. 805).

The phenomena of chromatolysis, or the changes taking place in the ganglion cells following pathological or experimental lesions of the axon, disappearance of the chromophilic granules, swelling of the cell body, displacement of the nucleus, etc., have occupied such a prominent place in the pathological field within the past few years, that the conclusions of Van Gehuchten upon these changes are of especial interest. These are as follows:

1. All pathological and experimental lesions of the axis cylinder of a motor neuron result in the process of chromatolysis in the originating cell body of this neuron, the duration and intensity of which being in direct proportion to the duration and intensity of the lesion.

2. Lesion of a peripheral nerve is not the only cause capable of producing chromatolysis. Such a phenomenon can follow in a variety of conditions, which fact must be borne in mind in the explanation of clinical conditions.

3. The section of the cellulipetal prolongation of a peripheral sensory neuron also results in chromatolysis of the cell of origin. This chromatolysis is more marked than that which is seen in a motor neuron. It is followed by the disorganization and disappearance of the corresponding cell.

4. The disappearance of the cells of the spinal ganglia, following section of their peripheral prolongations, is due not only to the lesion of these prolongations, but, of more import, it is due to the lack of trophic action which stimulation from without produces upon these nerve cells.

5. The ganglion cells in a nervous chain exercise the one upon the other a trophic action, the suspension of which produces a chromatolysis and disappearance of the corresponding cells.

6. The section or the lesion of a cellulifugal prolongation of the cells of the cerebro-spinal ganglia is not followed by profound chromatolysis, contrary to what is seen for the motor cells. In the present state of our knowledge this fact remains inexplicable. JELLIFFE.

84. THE EFFECT OF INANITION ON THE STRUCTURE OF NERVE CELLS. F. W. Barrows (Am. Journal of Physiology 1, 1898, p. 14, Proceedings, part 2).

The author studied the cells of the occipital cortex, spinal ganglia and cord in rats, comparing the cells of those which were well nourished with those that had been starved to death. Observations of the movements of the animals were also taken so as to compare the fatigue effects. The study shows:

(1) A decided shrinkage in the size of the cells and nuclei in the famished animals, averaging about 20 per cent., and a still greater shrinkage in the nucleoli.

(2) An evident exhaustion of the substance of famished cells, as shown by their faint staining with osmic acid and the notable absence of nuclei and nucleoli. The protoplasm of these cells shows a very fine vacuolation, not so marked as that described by Rosenbach for starving animals, and by Hodge for extreme fatigue. In the brains of famished rats the pericellular lymph spaces are considerably enlarged.

VOGEL.

85. SULLE ALTERAZIONE DEGLI ELEMENTI NERVOSI NELL'INANIZIONE (Alterations in the Nervous System due to Starvation). Lugaro e Chiozzi (Rivista di Patologia nervosa e mentale, 2, 1897, No. 9).

The authors starved animals for some time and then examined the nervous tissues by the newer methods. They conclude that in starvation the changes, occurring in the nervous tissues, are of slow development, that the loss of chromophilic substance is not marked, and is capable of prompt restitution until late stages of actual death are imminent.

In the animals studied great variations were observed. The posterior cells of the cord, of the spinal ganglia, the cortex and Purkinje cells were those most subject to change, while the large ganglion cells of the anterior horns suffered but slight alteration.

The chromophilic substances were affected in greatest measure, the achromatic substances suffering but little change.

The character of the changes is comparable in many ways to those induced by subacute or chronic poisoning by the metals, and the authors take the position, which appears to us rather fanciful, that the changes found in the nervous system may be induced by auto-toxic agents, which rapidly increase before death. JELLIFFE.

86. ANATOMICAL FINDINGS IN A CASE OF FACIAL PARALYSIS OF TEN DAYS' DURATION IN A GENERAL PARALYTIC, WITH REMARKS ON THE TERMINATION OF THE "AUDITORY" NERVES. A. Meyer (Jour. of Experiment. Med., vol. 2, 1897, p. 607).

The case of a paralytic, who, ten days before death developed a facial (complete) paralysis on the left side, is reported by Meyer, and the results of the autopsy given. Death ensued in a convulsion; post-mortem nineteen hours later. The facial nucleus was fixed in alcohol, 94 per cent. in part; the rest and the middle ear in formalin 10%. Clinically no evidence of ear trouble had been shown, but neither hearing nor taste could be examined owing to the dementia of the patient. A summary of the findings is as follows:

Medulla, right side, normal. Left side—nucleus of facial nerve showed the typical changes of reaction to a peripheral lesion as described by Nissl and others in experiments on animals.

There was no evidence of decussation of elements of the seventh nerve, the cells of the right nucleus being everywhere intact. The terminal nuclei of the eighth, both the dorsal and the ventral, showed well-circumscribed neuroglia-cell infiltration, whereas Deiter's nucleus was almost completely free. The central auditory cells were also slightly affected within the region of infiltration.

The case contradicts any decussation of the roots of the facial nerve, and speaks for a relative independence of the nucleus of Deiter's from the auditory nerve endings (contrary to the views of Bechterew). The facial palsy was caused by the condition within the internal auditory canal, i. e. a peripheral lesion, showing that the cells of one fibre system (the facial) can be involved by affection of another system (the auditory) with which it comes in contact. Two plates illustrate the article. STERNE.

87. SUR LA HISTOLOGIE PATHOLOGIQUE DE LA POLYNÉURITE DANS SES RAPPORTS AVEC LES LÉSIONS DE LA CELLULE NERVEUSE (On the Pathological Histology of Polyneuritis, etc.) S. Souklianoff (Nouvelles Iconographie de la Salpêtrière, 10, 1897, p. 347).

The author presents the clinical history and a complete cytological study of a case of multiple neuritis of probable alcoholic origin.

The methods of Nissl and of Marchi were used in the study of the nervous system. In the anterior horn cells there was marked central

chromatolysis; the nucleus occupied an eccentric position. In a few cases the nucleus was centric with some perinuclear chromophilic substance, but about this the chromatolysis was marked.

Following Marinesco's studies, the author shows that the lesions in the greater number of affected cells were similar to those seen after lesions of the peripheral nerves, and therefore secondary and not like those found when the lesion is a primary one, such as is seen in the case of the acute intoxications. The author interprets the lesion as primary in those cells, fewer in number, in which the nucleus remained central with perinuclear chromatolysis.

JELLIFFE.

88. SUR L'ABSENCE D'ALTERATION DES CELLULES NERVEUSES DE LA MOELLE ÉPINIÈRE DANS UN CAS DE PARALYSIE ALCOOLIQUE EN VOIE D'AMÉLIORATION (Absence of Alteration of the Nerve Cells of the Spinal Cord in a Case of Alcoholic Paralysis in Process of Amelioration). Dejerine et Thomas (*Comptes Rendus Hebdomadaires des Séances de la Société de Biologie*, 4, May 1st, 1897).

Dejerine and Thomas report a case of alcoholic paralysis of the inferior limbs with muscular atrophy; hyperæsthesia of the skin and muscles; talipes equinus; loss of patellar reflex; lesions of the cutaneous and muscular nerves of the lower limbs, consisting of empty nerve sheaths, and diminution in the number of large medullated fibres, without evidences of Wallerian degeneration. The anterior and posterior roots, the white matter and cells of the cord, appeared to be normal.

The writers think that the absence of the cellular lesions may have been due to the fact that the reaction of the cell body had ceased, the condition of the peripheral nerves being one of amelioration, although the power of motion was still much affected. The findings show, however, that in peripheral neuritis the nerves may be much altered without the presence of appreciable changes in their cell bodies. It is wise to maintain a certain reserve in judging of the importance of chromatolysis in the nerve cells, for, while it occurs after infectious processes or intoxications, it does not seem to have great significance. Goldscheider and Flatau have recently shown experimentally that pronounced cellular alterations may be found in animals which have presented no symptoms during life. These lesions are temporary, and the restoration of the nerve cell to a normal condition is very rapid. According to Goldscheider and Flatau, the corpuscles of Nissl are not of vital importance, and their physiological significance is unknown. Jacottet has noticed an absence of paralytic phenomena in animals intoxicated by different substances in which chromatolysis was found at the autopsy. Chromatolysis is, therefore, of interest cytologically, but is not of physiological importance.

SPILLER.

CLINICAL NEUROLOGY.

89. A CASE OF "LANDRY'S" PARALYSIS WITH AUTOPSY. T. Diller and A. Meyer (*American Journal of the Medical Sciences*, III, p. 104).

This case occurred in a female, fifty-three years of age, who was suddenly taken ill with paralysis of the legs, which was followed after three days by weakness of the arms, the bladder and intestinal canal. Three months later the patient died with bulbar symptoms, a gradual paralysis of the extremities having developed in the meanwhile. The patellar reflex was lost, but the sensations remained normal. Death was due to respiratory paralysis.

The microscopical investigation showed a slight sclerosis in the

crossed pyramidal tracts. The spinal roots were intact, and the author describes a pigmentation of the ganglion cells of the anterior horns. He concludes that the symptoms suggest a classification into two groups.

(1) In children, where the disease is termed acute anterior poliomyelitis.

(2) In older patients, when polyneuritis is accompanied by a special disease of the peripheral nerves, and Landry's paralysis with marked spinal symptoms.

JELLIFFE.

90. INFECTIOUS CAUSATION OF LANDRY'S SYMPTOM. Remlinger (Med. Week, 5, 1897, Nov. 5).

At the Biological Society of Paris, the author recalled a case previously reported by him of an acute ascending paralysis in which the streptococcus was detected in the spinal substance by cultivation, and was also found in stained sections.

In experimenting on the subject Dr. Remlinger has succeeded in producing acute ascending paralysis in a rabbit in which cultivation tests furnished evidence of the presence of the inoculated microbe in the cord. The microbe was a micrococcus derived from septic abscesses in a human patient. Dr. Remlinger thinks his successful production of ascending paralysis in this case is an additional argument in favor of the infective nature of its causation.

MITCHELL.

91. PARALYSIE ASCENDANTE AIGUE (Acute Ascending Paralysis). Hirtz et Lesné (La Presse Médicale, 5, 1897, p. 269).

The case described occurred in a woman of 22, beginning suddenly while she was in the third month of a so far normal pregnancy. The symptoms were first acute and then chronic. The disease lasted about four months, being then terminated by the death of the patient from bronchio-pneumonia. Two weeks before death, there was premature delivery, the child living but a few minutes. From this, however, the patient rallied quite well. There was complete paralysis of both lower extremities, and of the right upper extremity, with paresis of the left arm and of the back muscles. The respiratory muscles, and those of the face, tongue, palate, pharynx, and larynx remained intact. The paralyzed muscles showed no reaction of degeneration, but the tendon reflexes were lost. Sensibility was present, and the muscles of the lower extremities were very tender, and the seat of severe pains, increased on movement.

A clear liquid, withdrawn by lumbar puncture two hours after death, proved sterile. The brain and the peripheral nerves showed nothing abnormal. The vessels of both spinal pia mater, and cord proper, were greatly dilated, and their walls infiltrated with leucocytes. This was specially marked in the anterior horns about the ganglion cells. These latter were much altered, especially in the lumbar region, some having disappeared, others being shrunken and their nuclei displaced; others, again, hypertrophied. Above the cervical enlargement the cells were normal, and the vascular lesions disappeared in the bulb. There was no degeneration in the columns of the white matter.

C. L. ALLEN.

92. ZUR PATHOLOGISCHEN ANATOMIE UND AETIOLOGIE DER ACUTEN AUFSTIEGENDEN SPINALPARALYSE—LANDRY (On the Pathological Anatomy and the Etiology of Landry's Paralysis). L. Krewer (Zeits. f. Klin. Med., 32, 1897, p. 115).

Since 1893 the author has observed four cases of Landry's paralysis, in three of which he was enabled to perform autopsies. In his

three fatal cases there was a preceding history of alcoholism, one tubercular, and in all three the nervous symptoms were preceded by an attack of influenza. The pathological findings were those of an acute or subacute multiple neuritis and those of an acute myelitis.

The author concludes that Landry's paralysis should not be considered a disease *sui generis*, but should be regarded as an exceptionally severe form of polyneuritis, which involves not only the peripheral neuron, but also the spinal and the bulbar neurons, and that its chief etiological factor is some acute infectious disease, notably influenza.

JELLIFFE.

93 MULTIPLE NEURITIS FOLLOWING INFLUENZA. H. B. Allyn (Journal American Medical Association, 29, 1897, p. 152).

The author reports six original cases of multiple neuritis following epidemic influenza. From a study of his own and thirty cases collected from other sources his conclusions are as follows:

1. Influenza, like other infectious diseases, may be followed by neuritis and multiple neuritis.

2. One sex does not seem to be more liable to multiple neuritis than the other.

3. It occurs most frequently between the twenty-fifth and forty-fifth years; and appears during convalescence in a few days or two or three weeks after the influenza has subsided.

4. It may present sensory, motor, vasomotor or trophic symptoms, or all combined, but sensory and vasomotor symptoms are more prominent than in diphtheritic and some other cases of multiple neuritis.

5. The great majority of the cases recover, both as regards restoration of function and power as well as regards life. Five of the thirty-six cases collected in this paper died. In one of Bruns' cases the symptoms resembled Landry's paralysis, in the other there was paralysis of the tongue and throat. In Eisenlohr's fatal cases there was general motor paralysis with intense hyperæsthesia of the skin. In Ferguson's case the neuritis was visceral, and in Leyden's fatal case there was coincident disease of the cord.

6. Recovery does not usually take place under four weeks and may be delayed for months.

7. Treatment should consist first of absolute rest in bed. Anodynes must be given in sufficient doses to relieve pain, when that is a prominent symptom. Morphine hypodermically may be necessary, but may be often substituted for with advantage by cocaine. The antipyretic anodynes are insufficient in any safe dose if the patient has pains for many days. The salicylate of cinchonidin is distinctly valuable, especially when the pain is not of the greatest intensity. At a later stage potassium iodide and the bichloride of mercury in small doses are helpful. When the pain is in an extremity, firm pressure with a flannel bandage gives great comfort. Blisters over the painful nerve trunks when they are superficial are also valuable in relieving pain.

Close watch must be kept on the action of the heart and the character of the breathing. Most of the fatal cases die through paralysis of the diaphragm. The closest attention must be given throughout the course of the case to the nutrition of the patient and to the condition of the skin, especially over portions of the body where pressure occurs.

As far as possible the stomach should be reserved for food. Medicine in these cases acts better when given hypodermically, and the stomach is not so likely to be deranged. This caution applies especially to the giving of anodynes.

8. Finally, while he thinks diphtheria as a cause can be excluded in

the cases which he has seen, both from the absence of any clinical evidence of it in the patient or his surroundings and from the fact that diphtheritic neuritis is almost purely motor, yet he cannot exclude the poison concerned in the production of follicular tonsillitis—infectious tonsillitis, for sometimes this is associated with influenza, and it may produce as much headache, backache and prostration as usually characterize the onset of influenza itself.

SHIVELY.

94. MULTIPLE PARALYSIS FOLLOWING MEASLES. S. W. Morton (University Med. Mag., 9, 1897, p. 740).

The author reports what in all likelihood was a severe multiple neuritis following measles, in a child of two years and eight months. The possibility of mixed infection is not to be excluded, as deglutition was unusually painful and the entire course of the disease somewhat anomalous. The paralysis involved not only the extremities but also the muscles of the neck and those of phonation, articulation and deglutition. Four months after the beginning of the sickness the child had not yet fully recovered, although he looked well, and the knee jerks and faradic contractility of the muscles had returned.

PATRICK.

95. GONORRHOEA, MIT POLYNEURITIS COMPLICATA (Gonorrhœa complicated with Polyneuritis. E. Welander (Nord. Med. Archiv. N. F. 8, 1897, p. 26).

The clinical history is here given of a case of acute gonorrhœa with prostatitis and epididymitis occurring in a young man of twenty-one years of age. One month later symptoms of acute general infection appeared which soon disappeared, but superimposed upon these general symptoms there was a marked motor affection which persisted until the patient died of paralysis of the respiratory muscles and a purulent bronchitis. The nerves which were affected to the greatest extent were those of the face and the muscles of the limbs and trunks. The muscles of the bladder and intestine were paretic. There was no atrophy, the reflexes were abolished, and there was no reaction of degeneration. A microscopical examination (methods?) showed there were no changes in the spinal cord, nor of the nucleus of the seventh nerve. The peripheral nerves were in a profound state of degeneration.

VOGEL.

96. PRESSURE NEURITIS CAUSED DURING SURGICAL OPERATIONS. H. T. Pershing (Medical News, 71, 1897, p. 328).

The author reports several cases of injury to the brachial plexus and external popliteal nerve caused by carelessness in the position of limbs during long-continued operations. In summing up, he says: "The patient's arms should not be allowed to hang down, and care should be taken that during operation the weight of the body is as evenly distributed as possible. Keeping the patient in any constrained position should be avoided when not absolutely necessary, and the use of any mechanical contrivance for maintaining a desired position should be with due care to prevent nerves from being stretched or pressed upon. If neuritis does occur, the first indication is to secure absolute rest of the affected part during the early stage. At this time voluntary motion, massage, electricity, or any other excitant of muscle and nerve will do harm. When the pain and tenderness have subsided, counter-irritation, gentle rubbing and the galvanic current may be used to advantage. The faradic current often is harmful, and is useless except as a means of diagnosis or as a counter-irritant applied to the skin.

SHIVELY.

97. ZUR KLINIK DER FAMILIÄREN OPTICUSAFFECTIONEN (Clinical Communication concerning Family Diseases of the Optic Nerve). H. Higier (Deutsche Zeitschrift für Nervenheilkunde, 10, 1897, p. 489).

Higier describes two cases of optic neuritis occurring in brothers. An uncle of the patients was said to have had a similar affection. In one of Higier's cases the failure of sight began at the age of twenty; in the other at the age of twenty-seven. Central scotoma was found in both cases. This affection was described by Leber in 1871. The optic neuritis begins between the ages of thirteen and twenty-eight without demonstrable cause; there are central and paracentral scotomata for colors; the visual fields are not limited peripherally as in ordinary atrophy; nyctalopia is not rare; and the ophthalmoscope presents quite a characteristic picture. Although about twenty-five cases are reported, nothing is known of the nature and location of the inflammatory process. A neuropathic disposition is often noted. The peculiar fact that in the family form of neuritis only the macular fibres in the optic nerve are affected seems to be best explained by the greater functional activity of these fibres. This affection is not only a family one, but also hereditary, and is transmitted especially by females to their sons.

Higier speaks also of the syphilitic family optic neuritis, which should be distinguished from Leber's form, and is similar to the syphilitic retinitis. He describes also two cases of family optic atrophy which are unlike Leber's form. In the latter there is subacute neuritis axialis, in which, chiefly or exclusively, the macular bundle in the centre of the optic nerve is altered, and the temporal half of the papilla is atrophied; whereas in Higier's form the atrophy of the opticus is complete and chronic, and has an early development; the papilla is uniformly gray without any signs of neuritis, and the visual fields are concentrically limited without the presence of central scotomata. In addition to the cases of disease of the optic nerve dependent on malformation of the cranium, there is a family, possibly congenital, variety of optic atrophy entirely independent of such visible malformation.

Higier reports three cases of amaurotic family idiocy (Sachs) in one family, and compares this disease with the cerebellar hereditary ataxia. He refers to the presence of optic atrophy in hereditary and family cerebral and spinal diseases—a subject which he has treated at length in a previous paper. SPILLER.

98. A NEW SYMPTOM IN PERIPHERAL FACIAL PARALYSIS. Bordier and Frenkel (Medical Week, 5, October 1st, 1897).

The authors relate their observations of a phenomenon which they believe they are the first to mention, and which they think important for its prognostic value.

When a patient affected with severe peripheral facial paralysis is asked to shut his eyes, it will be found that the eye on the healthy side closes energetically, whereas, on the diseased side, there is but a very slight decrease in the width of the palpebral fissure, the globe of the eye, which remains visible to the observer, moving first upward, then slightly outward, the eyelid meanwhile finishing its movement of descent, the range of which varies in different cases with the degree of paralysis of the orbicular muscle.

The patient, in other words, cannot close his lids on the paralyzed side without at the same time deviating the globe of his eye upwards, and slightly outwards. If he is fixing some object before him, he is compelled to look away before he can contract his orbicularis palpebrarum.

In treating such cases with electricity, it was observed that improvement in reaction coincided with improvement in the closure of the eye. The symptom, therefore, gives us a means of following the progress of the improvement in these paralyses; recovery is near when the patient, who, at first, was obliged to turn one eyeball upward before he could bring its upper lid to its lowest possible point, begins to be able to shut his eye without deviating from the line of sight.

The phenomenon is then of help in three ways; first, as an aid in diagnosis; second, as a prognostic indication; third, as a measure of the progress of improvement.

First, it throws light on the diagnosis, seeing that it is not met with in cases of central paralysis. It is not necessary, however, to insist on this point, as there are other and simpler means of ascertaining the special form of a given case of facial paralysis.

Second, it establishes the prognosis, the presence of this symptom coinciding, as has been seen, with that of complete reaction of degeneration, it being totally absent when the reaction is only partial. When therefore contraction of the orbicularis is attended with deviation upwards of the eye, the paralysis may be considered as serious; but when contraction of the orbicularis can be effected without this deviation of the eye, the paralysis is not grave and will yield easily to treatment.

Third, it permits of following the progress of improvement, for the deviation of the eyeball also becomes less and less marked, in proportion as the reaction of degeneration tends to become partial; moreover, the patient is still liable to lower his upper lid without any deviation of the globe, when the reaction of degeneration consists simply in diminution of the faradic stimulability. MITCHELL.

99. DE L'ÉPICONDYLALGIE (Epicondylalgia). E. Rivière (Gaz. Hebdomadaire, 2, 1897, p. 685).

In 1896, Dr. Rivière in his thesis reviewed the history and status of the disease described by Benhard as professional neuralgia of the epicondyle, upon which Remak had already written. This neuralgia follows fatigue or over-use of the forearm, and is met ordinarily in violinists, or those who write a great deal. It is also located at the epicondyle, often in the muscular mass in the neighborhood of the epicondyle, and sometimes at the head of the radius.

The characteristic pains radiate from the epicondyle towards the external surface of the forearm, and extend even into the hand, of which they somewhat hinder the movements. The pains are lessened and disappear when the arm, and especially the hand, is in repose. Certain movements are especially painful, for example, those which include prehension with the extended arm. Pressure upon the epicondyle gives a lively pain well localized, although this is not the point at which the patients complain of spontaneous pain.

Like other professional neuroses, the affected muscles are somewhat impaired in their strength, and there is a special degree of feebleness in the movements of extension with prehension.

Rivière has not found that any special work predisposes to this affection. All occupations which necessitate the play of the epicondylar muscular mass may bring on this form of neuralgia, which he has observed in fencing masters, violinists, laboring men, coachmen, leather dressers and others. Like other professional neuroses, the treatment consists in rest and massage, and the earlier that these are applied, the better is the result. MITCHELL.

100. NEURITIS ISCHIADICA, NEURALGIA ISCHIADICA UND HYSTERIE (Sciatic Neuritis, Sciatic Neuralgia and Hysteria). Max Biro (Deutsche Zeitschrift für Nervenheilkunde, 11, 1898, p. 207).

Biro presents some diagnostic points between inflammation, neuralgia, and hysterical disease of the sciatic nerve. In neuritis the Achilles tendon reflex is absent, and altered electric reaction and muscular atrophy are noticed; these signs are not present in neuralgia. Painful points (*points douloureux*) are present in neuralgia, and are rare in neuritis. When no symptoms of inflammation, no disturbance of sensation, other than the *points douloureux*, are noticed, the case may more properly be considered one of neuralgia than one of neuritis, but from the perusal of the paper it is evident that Biro does not regard the distinction between neuritis and neuralgia as very sharp. The Achilles tendon reflex has not received the attention it deserves; often it disappears in disease before the patellar reflex (tabes, diabetes). When the sciatic nerve is only slightly affected, the Achilles tendon reflex may not be altered, and every doubtful case in which this reflex is present is not one of neuralgia, but its absence always indicates neuritis. Hysteria not infrequently simulates true sciatica, but in hysteria the painful points are absent, or are not limited to the course of the nerve, passive movements are not painful, Lasèque's sign (flexion of the extended lower limb on the trunk causing pain) is not present, the pain is seldom confined to one extremity, the muscles do not atrophy, no change is noted in the electric reactions, and the tendon reflexes are not absent. Hysterical sciatica occurs at a period when true sciatica is uncommon (before the twentieth year), is more frequently observed in females, and may be associated with other signs of hysteria. No theory satisfactorily explains the scoliosis of sciatica, and many of the so-called causes are doubtful.

SPILLER.

PSYCHOLOGY.

101. AFTER-SENSATIONS OF TOUCH F. N. Spindler (Psychological Review, 4, 1897, Nov. 6).

Frank N. Spindler experimented on five subjects, applying weights ranging from 25 grms. to 1,000 grms. to the back of the hand. He found it absolutely impossible to have the results absolutely accurate, as the after-sensations fade always gradually. He sums up the results as follows:

(1) The minimal time of stimulation which will yield an after-sensation of the kind under investigation is about 5 seconds, with a pressure of 150 grms.

(2) The relation between the duration of stimulation and the length of the interval which elapses before the appearance of the after-sensation is very irregular. The intervals increase up to stimulations of about 3 minutes and then again decrease.

(3) The duration of the after-sensation increases with the duration of stimulation, though without any discoverable regularity.

(4) The longest duration of after-sensations is given by pressures of from 150 to 500 grams. Above and below these limits of pressure the duration decreases.

(5) In quality the after-sensations are very variable. The writer could discover no waves in his own after-sensations, but only a steady, persistent feeling of contraction. Other subjects experienced waves of heat, of pain, etc., but they also in most cases felt a steady, persistent underlying touch on contraction sensation, lasting through the dull aches, the smarts, and the heat or cold.

CHRISTISON.

102. THE PSYCHOLOGY OF TICKLING AND LAUGHING. G. S. Hall and A. Allen (*American Journal of Psychology*, 9, 1897, pp. 1-42).

The authors received reports from nearly 3,000 persons in response to a syllabus sent out. The analysis shows that in exceptional cases the first symptom or source of laughter may take place in almost any part of the body, but it is most frequent in the eyes and next in frequency at the mouth. The eyes become brighter, smaller and oscillate, and the mouth opens, stretches and curves upward, but sometimes downward. In some few cases the laugh begins with dimples in the cheeks; in others a movement of the muscles just below the ears; in others a throwing back of the head.

Subjectively, too, the "funny feeling" may begin in the stomach, throat, head, diaphragm, face etc. Sometimes beauty is evoked or increased, or ugliness is produced. The eyes are sometimes open, sometimes shut, sometimes grow dull, both lids may tremble and balls twitch. They may grow rigidly fixed or roll wildly, may be turned upward and inward and are often suffused with tears. The mouth, too, may take almost every variety of position, as does the eyes, and almost any sequence may be inverted.

In the height of laughter individual peculiarities become multiplied and emphasized. Two returns described laughter so intense that death from ruptured blood-vessels ensued.

The onset may be very gradual or instantaneous and explosive; and the omitted sounds may be almost any kind of noise, although *he*, passing over to *ha ha*, are the usual. Some sob.

The after effects of a hearty laugh were described as exhaustion, heavy breathing, fatigue, shame, weakness, depression, soberness, sadness, relief, weakness localized in various parts of the body, the deep sighs, giddiness, perspiration, headache, stitch in the side, soreness, thirst, sweating, chills, sleepiness, uncontrollable movements, nausea, tears, fear of impending disaster, breathlessness, etc. On the whole, Dr. Hall remarks, the laugh is not unlike an epilepsy from the aura, at which stage it may be checked, to the subsequent exhaustion. In a number of cases laughter was evoked by news or sights really sad.

Dr. Hall refers to Brücke's experiments, showing that when the head is thrown back, shoulders up, and the body generally is bent backward, the blood tends to flow from the arteries, where pressure is high, into the veins, where pressure is low, and thus, he concludes, that "if laughter is more often associated with the later position, and crying with the former, this would go far to account for the subjective difference between the two and would connect the relief of a laugh with the remission of arterial tension. In the majority of adults it begins with the highest level in consciousness and the finer muscles, and passes down to lower levels and earlier developed musculature, although sometimes in children this order is exactly inverted."

From the returns children were found to be the most ticklish, as follows: Soles of feet, 117; under the arms, 104; neck, 86; under the chin, 76; waist and ribs, 60; cheeks, 58; knee, 25; down the back, 19; behind the ears, 15; palms, 14; corners of mouth, 8; breast, 8; nose, 7; legs, 5; elbows, 3; lips, 3, etc. Some of these children mentioned several places and so appear several times. Two small children are so ticklish they scream with laughter if touched. Mention of the word "soles" would, in some cases, cause slight tickling, and the mere pointing the finger in 107 cases. When near the cry-point, some children, otherwise very ticklish, are like stone to every stimulus. Some are ticklish only near bed time or when very tired. Sixty clearly marked cases were ticklish when "hanny" or "unwell," or "after a good meal," "when perfectly well," etc., and their susceptibility, through the different senses, varied greatly.

CHRISTISON.

THERAPY.

103. A CLINICAL LECTURE ON SOME CASES OF INJURY TO THE ULNAR NERVE. Bennett (Clinical Journal, Nov. 10th, 1897).

In this lecture there are four points worthy of mention.

1. In nerve suture it is not necessary to remove all of the bulb which may have formed on the proximal end. Indeed, it is better to leave part of it as the stitches will hold better than in normal nerve tissue.

2. Sensation may improve within twenty-four hours, to be again lost after two or three days, without at all affecting the prognosis for ultimate recovery.

3. When, after an injury to a nerve the pain spontaneously, or on pressure, shoots upward toward the origin of the nerve the prognosis is much more serious than when it shoots toward the periphery.

4. Many months may elapse after suture before the first sign of improvement, and yet ultimate recovery be almost perfect.

PATRICK.

104. TREATMENT OF SCIATICA. A. Marty (Medical Week, 5, 1897, July 30th).

The author in his thesis relates that two cases of sciatica which had resisted every variety of medical treatment, were cured by Dr. Gérard-Marchant by means of an operation which consisted in tearing apart the fibres of the nerve with a blunt instrument. This treatment had previously been employed by Dr. Delagénère, who denuded the nerve and teased the nerve fibres with a pair of hæmostatic forceps, his object being to destroy any small veins which might exist in the interior of the nerve and thus prevent any stasis in them. It seems possible enough that the successful results were as much due to the rest in bed and the immobility of the limb which the operation made necessary, as to the operation.

MITCHELL.

105. DE LA CURE RADICALE DU MAL PERFORANT PAR L'ELONGATION DES NERFS PLANTAIRES. (Nerve Stretching for Perforative Ulcer). Chipault (La Médecine Moderne, April 7th, 1897).

The author reports on a radical cure for perforating ulcer of the foot by elongation of the plantar nerves.

After discussing various other surgical means of treatment of nervous diseases—lumbar punctures and reduction by operation of the deformity of Pott's disease, M. Chipault considers further the question of perforating ulcer and the success attending this treatment. Amputation is not always successful, as the ulcer is apt to return upon the stump. In such case a second amputation becomes necessary. In fact, a perforating ulcer is only a manifestation of a number of different affections of the nervous system.

The author states that he has performed the operation on the plantar nerves for this disease seven times; five of the operations were done more than two years ago. He advises that the operation be performed at some distance from the seat of the disease, selecting, according to the case, the plantar nerve below the internal malleolus; the muscular cutaneous above the external malleolus; or the external saphenus at the margin of the tendo-achillis.

Of the seven cases operated upon, but one was not cured. The causes were various—locomotor ataxia, frost bite, traumatic neuritis. All the cases remained healed.

The same operation was proposed by Dr. Weir Mitchell, two or three years ago, for erythromelalgia, and successfully performed in several instances by Prof. W. W. Keen and Dr. T. G. Morton.

MITCHELL.

Book Reviews.

THE NERVOUS SYSTEM AND ITS DISEASES. A Practical Treatise on Neurology for the Use of Physicians and students. By Charles K. Mills, M. D., Professor of Mental Diseases and of Medical Jurisprudence in the University of Pennsylvania, etc.

DISEASES OF THE BRAIN AND CRANIAL NERVES With a General Introduction on the Study and Treatment of Nervous Diseases. With 459 illustrations. J. B. Lippincott Co. Philadelphia, 1898.

The author has been known, here and abroad, as one of the ablest among American neurologists. His scientific work has always been characterized by exceeding thoroughness and sound judgment. It was, therefore, a foregone conclusion that a book from Dr. Mills' pen would be distinguished by these same high qualities; but, even so, it came as a surprise to us to find that it is altogether the most ambitious effort in the line of neurological book-making. It exceeds in size the corresponding volume of Gowers and the monograph of Von Monakow, and in excellence of its contents, we hasten to add, it is surely the equal of either of them.

Of its 1,056 pages, 256 are devoted to a general Introduction to diseases of the nervous system, and the remaining (nearly 800 pages) to diseases of the brain and of the cranial nerves. We do not see why the author and publishers have not called this "Volume 1," since it is to be followed by another, "which shall include the remaining diseases of the nervous system, insanity, and the medical jurisprudence of both nervous and mental diseases."

The first chapter is called (modestly) a sketch of the nervous system; it contains, in fact, a sufficient account of the development and of the architecture of the nervous system. The embryological data will be particularly welcome to English-speaking readers, for they have been omitted too often from English text-books. The statements made are accurate, and, as far as it is at all possible, illustrations have been utilized to make the subject matter clear to the student. But it is to be feared that the student will not be grateful to Dr. Mills for the introduction of the new cerebral terminology of Wilder and Gage. While recognizing the full merits of this new nomenclature, and appreciating the benefits conferred upon the comparative anatomist and the comparative embryologist, the truth is, the student of neurology does not need it. He can get along well enough with the old terms. The prosocoele, the mesocoele, the epicoele, the metacoele, and all the other "coeles," are not a great advance upon the good, old-fashioned designations for the ventricles; nor does one take kindly to the various "mononyms" (the translation of this term is found in the foot-note on page 47). "Postoblongata" is not particularly euphonious, nor in keeping with the spirit of the language, and it will be a long while before "Metapore" eclipses the "foramen Magendie." Life is proverbially short, and neurologists care less, at present, about the cavities and holes of the brain (the "coeles" and the "pores") than they do about tracts and cells; and since Van Ge-

huchten has recently blessed us with "tautomeral," "heteromeral" and "hecatomeral" cells, we shall be kept busy enough with these. Mills has evidently felt some hesitation in introducing a section on nomenclature, and we find that in the middle of the book he himself prefers to speak of "lesions of the ventricles," and not of the "coeles." We have no doubt that in the course of time some of these names will be adopted by general consent; but it will be well along in the next century before the system, as a whole, will come into use. It has been suggested that children should begin the study of brain anatomy. The plan is a good one with reference to this nomenclature; the only way to acquire it is to acquire it early in life, when the cortical cells are ready for the reception of any and all auditory impressions.

In the section on general architecture and physiology of the brain, the sensory tract is mapped out with great detail. Comparative tables like the one on page 105 add greatly to the value of this treatise. The little that is known of the chemistry of the brain and of the chemical processes accompanying nervous action is stated clearly. Altogether this first chapter bears testimony to the author's scholarly attainments.

The second chapter opens with a brief consideration of the general pathology and etiology of nervous diseases. These are included under a few general heads, such as, Developmental Malformations; Inflammations; Degenerations; Tumors and Adventitious Products; Disease of Blood-vessels; Vascular Disturbances; and Functional Disorders. The remarks made under each heading help to establish general principles of neuro-pathology, of which our physicians and students are so much in need. Speaking of degeneration, Mills claims "that primary degeneration may be due to embryonal arrest, or it may be infectious or toxic." The first half of this statement is in keeping with views recently expressed by the present writer, and embodies a fact that is often disregarded. Inflammation is described either as an exudative (serous, fibrinous or purulent), or as a proliferative process, the latter being dependent upon syphilis and other infectious diseases.

The etiology of nervous diseases is considered very fully, to the inclusions of seasonal influences and other Philadelphia specialties. It does not, however, appear to be the season alone which has this curious influence in developing infectious diseases in those predisposed by neuropathic constitutions; for we learn that the tone of the nervous system becomes lowered "in the spring, as the result of severe and prolonged labor" (which is not a peculiarity of the seasons), "or as the effect of cold and exposure." In passing, it may be of interest to note that in New York chorea, for instance, according to the statistics of several clinics, begins as often in August as in the spring months. The consideration of the microbic origin of nervous diseases, and the relation of the infectious processes to them, is thoroughly in accord with recent doctrines. The symptomatology and methods of investigations in nervous diseases are discussed in concise form within the compass of 36 pages, and we fail to find any serious omissions. In view of this, electricity is treated too liberally, being spread over 31 pages. Yet, these are matters of which too much should not be made. Moreover, 47 pages are devoted to general therapeutics. How thoroughly "thumbed" these pages will be, filled as they are with an account of almost every therapeutic agent ever recommended, with a capital table of the "untoward effects of some of the more potent drugs," with innumerable formulæ, with a recommendation of Nuclein Therapy, and with a cautious mention (fortunately not an endorsement) of Cerebrin and "Testicular Therapy." The "table of doses of the potent and newer drugs used in neurological practice" is invaluable, and we record with pleasure that in a foot-note the author does full justice to the late Dr. Sequin's work in this especial field.

With Chapter III. begins the description of the various diseases

of the brain. The arrangement is novel in many respects. Diseases of the membranes are discussed, together with those of the sinuses and the veins of the brain, and the chapter closes with an account of encephalic malformations and aberrations, including chronic hydrocephalus. Tubercular and cerebro-spinal meningitis are discussed under the heading of Lepto-Meningitis, where they properly belong. The bacterial origin of the various forms is given due weight. In the treatment of meningitis Mills is inclined, on the whole, to be conservative; "Lanphear, Keen and Senn have advocated the surgical treatment of tubercular lepto-meningitis." But, does Dr. Mills recommend it?

The next chapter is one of the best in the book. The minute anatomy of the cortex precedes the discussion of cortical localization, and this is followed by a study of the lesions and diseases of the larger ganglia, by an account of cranio-cerebral topography, and by a complete statement of the manner in which a post-mortem examination should be made, in order to verify the exact site of a lesion. In discussing cortical localization, Mills, naturally enough, adheres to his own well-known views regarding an independent sensory area, but makes reference to the views of his opponents. He also decides that the evidence is, on the whole, in favor of setting apart a special naming centre, and he would place it in the third temporal convolution. The encyclopedic character of the book is brought out in this chapter by the publication of many facts which the student of neurology is anxious to have, but would not know where else to find; such as the average brain weight of the insane, the recorded "brain weights of eminent men," and the like.

Encephalic circulation and the vascular disturbances and diseases of the brain are described in a masterly manner in Chapter V. It is interesting to compare the table of differential diagnosis between hemorrhage, thrombosis and embolism, as given by Mills, with one published almost simultaneously by von Monakow. In the main they agree. Mills lays stress upon the pupillary symptoms; Von Monakow scarcely mentions them; but the latter refers in greater detail to the psychic phenomena preceding and following the attack, which Mills treats less elaborately.

The section on tumors of the brain, encephalitis, cerebral paralysis of children and aphasia show the author's intimate acquaintance with each special subject. The various forms of encephalitis are given with as much detail as the present state of our knowledge permits; but in this, as in almost all other subjects, the reader will find that the author has kept a close watch on medical progress, without exhibiting an undue fondness for theories that have not been sufficiently substantiated. In writing upon aphasia, Mills is actuated by a spirit of fairness to those who do not accept his frequently expressed views. He reiterates his belief in a special motor graphic center, but, at the same time, does full justice to the views of Dejerine, Wernicke, and others. The clinical relations of aphasia to other paralytic phenomena are brought out more distinctly than by the majority of writers on aphasia.

In Chapters VII. to X. the affections of the special senses are described in connection with all other cranial nerve diseases. We say advisedly that we know of no other book in any language in which this part of the subject has been treated with equal thoroughness. Diseases of the auditory and optic nerves are explained in such a way that, we trust, oculists and aurists will regard this part of Mills' treatise as a work especially designed for them. We commend once more the satisfactory manner in which the author has described these diseases always prefacing the description of disease by a thorough account of the anatomy of the peripheral sense organ or nerve and its central connections.

But there are other features about this work which will stamp it as one of unusual excellence. The style is terse and clear throughout. The illustrations are good and abundant, and there is a bibliographic index which might well be called "The Index Medicus" for neurologists. We realize the stupendous labor bestowed upon the preparation of this work, and it is evidently planned and executed with the utmost deliberation. We rejoice heartily in the publication of this treatise, which will be not only a lasting credit to its author, but will help to place the study of nervous diseases upon a higher plane. We shall await anxiously the appearance of the second volume.

B. SACHS.

THE BULLETIN OF THE OHIO HOSPITAL FOR EPILEPTICS. Gallipolis, Ohio, January, 1898.

To Ohio belongs the credit of having been the first State in the Union to establish a special hospital for epileptics. The management shows a progressive spirit in attempting, over and above the mere care and treatment of the inmates, to conjoin with the good work done there scientific investigation as to the causes of epilepsy. It must be through long-continued effort of pathological chemists in well-equipped laboratories that important discoveries will be made, which some day will elucidate the problems of epilepsy and indicate better means of cure than we now possess. We are glad to see that the Ohio institution is fully alive to the opportunities afforded it by the vast material at its disposition. The work of the special pathologist, Dr. Ohlmacher, as presented in this Bulletin, is to be commended for thoroughness and precision. The first forty pages are taken up with the minute details of autopsies upon six patients. He paid especial attention to the condition of the thymus gland in these cases, finding four instances of persistent or enlarged thymus, and makes the relation of thymic hyperplasia to epilepsy the subject of a second article in the same number of the Bulletin. In his conclusions Dr. Ohlmacher says that these morphological anomalies of the thymus observed in his cases of epilepsy, and which have also been noted in thymic asthma, thymic sudden death and possibly exophthalmic goitre, will be found to have something more than an accidental relation. We trust that he will continue his researches in this direction, and that others engaged in similar work will follow up the new clue in the attempt to unravel the secrets of this obscure condition.

Dr. Rutter, the superintendent, devotes a few pages of the Bulletin to "Colony Care of the Epileptic." We are afraid that from the first the management of the Gallipolis institution has not kept in mind the true *Colony* system. Beginning with but 125 acres of land there have been added only 125 acres since—an acreage too small for the number of patients already in the hospital. The plan adopted was originally that of a pavilion-asylum—not a colony at all. It consisted of stone buildings, each for fifty patients, symmetrically arranged about an administration building and connected by tunnels with central power house, kitchen and bakery, and flanked by two congregate dining rooms, one for each sex. The group was planned for 1,000 patients. While there has been some modification of the original plan, Dr. Rutter's description reads as follows:

"The buildings will then consist of eleven residence *cottages* with from fifty to seventy-six beds each; one laundry-cottage for seventy-five resident patients; one *cottage* for the insane with a capacity of two hundred," etc.

The italicization of the word "cottage" by the reviewer is merely to indicate an apparently wrong use of the word. Surely one cannot designate as cottages buildings accommodating from fifty to two hun-

dred patients! The same misinterpretation of the meaning of cottage is evident in the misconception of the term colony by Dr Rutter. The Ohio institution is not a colony. It is a special hospital for epileptics on the pavilion plan, and seems to present none of the features which distinguish Bielefeld and the institution at Sonyea, N. Y., as colonies in the true sense of the term. The Gallipolis Hospital for Epileptics does not differ essentially from the asylums at Toledo or Ogdensburg as regards structural arrangement. We emphasize this matter because there is a vast difference between an asylum or pavilion-hospital and a village-community or colony. We emphasize it not with a spirit of carping criticism, but with the hope that the management of the Ohio institution may yet be able to modify the scheme of its development in accordance with the ideals of the colony system. F. PETERSEN.

BOOKS RECEIVED.

"A Compendium of Insanity," illustrated, by John B. Chapin, M.D., LL.D. W. B. Saunders, Phila.

"The Surgical Complications and Sequels of Typhoid Fever," by William M. Keen, M.D., LL.D. W. B. Saunders, Phila.

"Atlas of Methods of Clinical Investigation with an Epitome of Clinical Diagnosis and of Special Pathology and Treatment of Internal Diseases," by Christfried Jakob, M.D.; translated by Augustus A. Eshner, M.D. W. B. Saunders, Phila.

"A Modern Pathological and Therapeutical Study of Rheumatism, Gout, Rheumatoid Arthritis and Allied Affections," by E. L. Gros, M.D.

"Mechano-Therapy and Resistance Movements in the Treatment of Heart Disease," by Thos. S. Dowse, M.D., F.R.C.P. John Wright & Co., Bristol, Eng.

THE
Journal
OF
Nervous and Mental Disease

Original Articles.

A CASE OF LANDRY'S PARALYSIS.

By W. L. WORCESTER, M.D.,

Assistant Physician and Pathologist, Danvers Lunatic Hospital.

The following case of acute and fatal paralysis, which apparently should be classed with those first described by Landry, presents some peculiar features which seem worthy of record, apart from the comparative rarity of cases of this kind:

Caroline K., a widow, aged 40, was admitted to the Danvers Lunatic Hospital, April 14, 1896. According to the statements of her sister and physician, she first began to show symptoms of mental derangement about the middle of December, 1895. She first imagined that she was pregnant, though she had ceased to menstruate three years before, and consulted a number of physicians on this account. Subsequently she imagined that she had blood poisoning from a dead foetus. She soon took her bed, and kept it until about March 1, at which time, at the urgent command of her brother, she began to sit up during a part of the day, and walked out a little. On hearing of the death of Governor Greenhalge of Bright's disease, she concluded that such was her complaint, and resumed her bed. She complained of violent abdominal pains, and often made a great outcry. Three or four weeks before her admission, her urine was noticed to be "bloody," and an examination at this time showed the presence of albumin and casts, which had not previously been found.

About four weeks before admission she began to complain of numbness in her hands. She manifested no difficulty in using them until about a week ago. On the 9th inst. she spilled some medicine she was trying to take, and from that time on used her hands but little. Two days later she seemed completely paralyzed in all the extremities. The paralysis was considered by her physician to be merely a hypochondriacal delusion, and she was said to have been noticed to make movements when not watched.

Among other sedatives, small doses of sulphonal and trional had been given, but, according to her physician's statement, he had prescribed nothing of the sort for some weeks before her admission.

The patient had suffered, some years previously, from paralysis of the left facial nerve, from which she had only partially recovered.

No history of insanity in the ancestry was obtained, but a son of the patient is an epileptic idiot.

On admission the patient was found to be a slender woman, not markedly emaciated. No evidence of disease of the thoracic or abdominal viscera was found on physical examination. There was a moderate degree of paralysis of the left facial nerve; the eye could not be completely closed, and the innervation of the mouth was inferior to that of the right side. Otherwise, the movements of the facial muscles seemed unimpaired. There was apparently almost complete paralysis of all the extremities. On passive motion they fell into positions determined entirely by gravity. In all the extremities the paralysis seemed to increase in intensity toward the trunk. The fingers and toes could be moved a little; the hands and feet very slightly; arms and forearms, legs and thighs, not at all. In respiration the diaphragm alone was active.

The pupils reacted normally to light. Knee jerks could not be elicited. There was no response to tickling the soles of the feet.

The reaction to the faradic current was very slight in

the hands and forearms, feet and legs, of both sides. Reaction was less on the right than the left side. The ulnar and median nerves were less excitable than the muscles supplied by them. No reaction at all could be obtained by the full power of the (rather weak) battery in thighs, upper arms or pectoral muscles. The galvanic battery was found not to be in working order.

There was great impairment of sensation, corresponding in distribution to the motor paralysis. The head and face seemed entirely unaffected. On the hands and feet touch, pain and temperature were fairly well felt and localized; on the forearms and legs, there was very marked impairment of all forms of sensation; on the upper arms and thighs, only very firm pressure could be felt, and a pin could be thrust through a fold of the skin without her appearing to notice it at all, although, when questioned, she said it felt like something sticking in. Over the abdomen, transfixion of a fold of skin felt like something hot.

The visual fields were not narrowed, and there seemed to be no impairment of any of the special senses.

Mentally, the patient seemed rather apathetic. She answered questions relevantly, and, so far as could be determined, correctly, but seldom spoke otherwise. Memory appeared rather poor. There was, at the time of the examination, no appearance of hypochondriacal delusions, or of a disposition to exaggerate her symptoms.

A specimen of her urine attracted attention at once by its peculiar appearance. It was of a claret color, very different from that usually imparted by blood; acid in reaction; contained a trace of albumin and a few hyaline casts.

No change was noticed in her condition during the night of the 15th. At 9.30 of the morning of the 16th she asked for the bed-pan, in order to have a movement of the bowels. Immediately afterward the nurse noticed a change for the worse. A physician was called at once, but when he reached the ward respiration had ceased, although the heart was still beating.

At the autopsy, five hours after death, no lesion of the nervous system could be discovered. The only morbid appearances found were slight emphysema and a catarrhal condition of the gastric mucous membrane.

The results of the histological examination were as follows:

Cerebrum.—The only changes noted were in the large nerve-cells, characteristic of the excitable region of the cortex. A large proportion of these presented a swollen appearance, giving the impression of the deposit of a morbid material in their interior. In specimens stained by the Nissl method the marking was confined to the surface and the processes of the cells. Both in these specimens and in those hardened in bichromate solution and stained with carmine, the nuclei of these cells were displaced, either into one of the processes, or to the surface of the cell, in which latter case they often projected beyond the surface. These changes are familiar enough to all who have done such work with the Nissl method, and are observed in a variety of morbid conditions. No morbid alterations were detected in the other regions of the brain examined, and the nerve fibres, in specimens stained by Pal's method, appeared normal.

Medulla Oblongata.—Sections at different levels, stained by Pal's and Van Gieson's methods, appeared normal.

Spinal Cord.—Sections from the lumbar enlargement, stained by Nissl's method, showed, in a considerable number of the large cells of the anterior horns, an apparent breaking up of the colored substance in the interior of the cells into fine granules. A few cells presented changes similar to those described in the brain. Side by side with these were numerous cells that appeared entirely normal. Sections from various parts of the cord stained by the Marchi method showed a few degenerate fibres, but not enough to account for any serious symptoms.

Specimens hardened in bichromate, and stained with

carmine and by Pal's and Van Gieson's methods, presented no abnormal appearances.

Peripheral Nerves.—The sections of the nerve roots made in connection with those of the spinal cord appeared normal by all the methods of staining employed. Specimens from a cord of the lumbar plexus and the right radial nerve were prepared by Marchi's method. A few varicose nerve fibres stained faintly with the osmic acid, but there was no appearance of segmentation of myelin.

Specimens from the radial nerve and several muscular branches were preserved in solution of osmic acid and dissociated. Many of the fibres showed much varicose swelling, but no segmentation of myelin was found.

Kidney.—The only abnormality found was an apparent flattening of the epithelium of the convoluted tubules.

We have here a rapidly developing motor and sensory paralysis, the former predominating, affecting the muscles of the trunk and extremities, and sparing those of the head and face, proving fatal, apparently, by arrest of respiration. Concurrently with this was observed a peculiar color of the urine, attributed by her physicians to blood. The urine contained no blood corpuscles at the time of my examination.

It seems to me improbable that this is a mere coincidence. The history of the case is suggestive of a toxic process, and it seems probable that the change in color of the urine was indicative of a morbid condition of the blood. I at once suspected porphorinuria from sulphonal intoxication, but was unable to learn that she had taken more than a few doses of sulphonal and trional, some time before the symptoms referred to made their appearance.

The anatomical lesions found, though they may, perhaps, be brought into connection with the symptoms, do not seem to me to account for them satisfactorily. From the fact that stimulation of the muscles supplied, for instance, by the ulnar nerve, produced stronger contractions than stimulation of the nerve itself, it seems evident that

the lesion could not have been confined to the central nervous system, but that the nerves must, at least, have participated in it. The fact that both motion and sensation were involved over the same area would point in the same direction. Yet no unequivocal morbid appearances could be found in the nerve roots or their peripheral distribution.

The question of classification now presents itself, and is, to me, a rather perplexing one. So far as I have been able to discover, the only commonly recognized form of nervous disease to which the case can be referred is that to which reference has already been made—the acute ascending paralysis described by Landry. The description of this disease given in all the text-books to which I have access agrees with the definition given in Gould's Medical Dictionary, which I will quote: "A form of atonic paralysis, described by Landry, characterized by a loss of motor power in the lower extremities, gradually extending to the upper extremities and to the centres of circulation and respiration, without characteristic sensory manifestations, trophic changes, or variations in electrical reaction."

It is at once evident that, if all parts of the foregoing definition are considered essential, my case is excluded by the very pronounced impairment of both sensation and electrical excitability.

Walton, in a paper published in the *Boston Medical and Surgical Journal*, December 26, 1895, takes the ground that Landry's paralysis is really a multiple neuritis. He analyzes the published cases in regard to sensation and electrical reaction, and finds that, among 122 cases, disturbances of sensation were reported in 74, and that only in 16 was it definitely stated that sensation was unimpaired.

In a large number of the reported cases the electrical reactions were not ascertained, but reaction of degeneration was found in a considerable number. Out of the 122

cases analyzed by Walton, only five were found in which sensation and electrical reaction were both stated to be normal. It is, therefore, apparent that if the above definition is to be considered authoritative in all particulars, a large proportion of the reported cases are incorrectly assigned to this disease.

Walton proposes the following description:

"Landry's paralysis is an acute, toxic disease, characterized by rapid loss of power in the lower extremities, trunk, and, to a less degree, in the upper extremities, affecting also the vagus and phrenic, sometimes other cranial nerves. The affected muscles are lax. Pain, paræsthesia, anæsthesia and tenderness are generally present in varying degrees, though, in some cases, sensory disturbances are wanting. Death follows in more than half (64%) of the cases. Recovery, when present, is very slow. The reflexes, deep and superficial, are lost at an early stage; wasting and degenerative reaction appear if the patient survives. The process is a toxic affection of the peripheral nerves (neuritis), cord and brain, the former being the essential and persistent lesion."

My own case would come, easily enough, under the foregoing definition. It is true that the lesions characteristic of multiple neuritis were not found in the nerves, but the electrical reaction, already mentioned, proves conclusively, to myself, that their function was impaired, and it is quite possible that the case had not lasted long enough for the development of the characteristic lesions.

That the case was of toxic origin can, I think, hardly be doubted. The nature of the poison is, unfortunately, obscure, but it seems reasonably certain that it was in some way connected with the peculiar color of the urine, which made its appearance at about the same time with the paralysis. It seems altogether unlikely that this is the first or last case of this kind, and it is to be hoped that some future observer will be more fortunate than I in this particular.

GLIA AND GLIOMATOSIS.

By SIMON FLEXNER, M.D.,

Associate Professor of Pathology in the John Hopkins University.

Abstract of remarks made before the Philadelphia Neurological Society, Feb. 28th, 1898.

Embryologists and histologists have by their recent work prepared the way for a reconsideration of the pathological features relating to the neuroglia. They have shown that the "connective tissue" of the brain—to use the term in its old signification—is an ectodermal structure, that its blastodermic precursor is the same as for the nerve cells; that it is less profoundly specialized, less highly differentiated than the nerve cells, and that it subserves a more humble purpose than these. The histologist encounters neuroglia in its several phases as it passes from its embryonic to its adult state, while the pathologist meets with it in the developed body under hardly less protean conditions. A complete knowledge of the histology and histogenesis of neuroglia must embrace all the conditions—normal and pathological—which have been observed. To the pathologist such a complete knowledge must be of the first importance, since upon it will depend his conception of the origin and the nature of a group of new growths occurring within the central nervous system.

In order that the pathological facts which I wish to bring before you may be presented in the light in which they appear to me, I shall be obliged to ask your attention to a brief account of the histogenesis of neuroglia, which, for our present purposes, can be best investigated in the spinal cord. The cells destined to become glia are derived in the embryo from the medullary plate, and are at first of the same value as the elements which eventually

produce nerve cells. The possibility that cells of other values, leucocytes and endothelial cells, may, at a later period, become interpolated between the elements of ectodermal origin, and, under certain pathological conditions, act as tissue formers, while admitted by so good an authority as Ramon y Cajal, have been shown to be improbable by the researches of Schaffer and v. Lenhossék.

The glia cells—or, to use the term proposed by Fish in this country and v. Lenhossék in German, astrocytes—take their origin in the lowest and highest vertebrates from the ependyma cells, which are now known to belong to the supporting cell structures of the cord. In certain low forms (myxine, amphioxus) the ependyma cells represent the total of the supporting cells, while in the higher forms greater or less numbers of astrocytes supply the chief framework of the organ. In mammals, and especially in man, astrocytes may, it is considered by some, develop from a less highly differentiated cell than ependyma cells, which yield them exclusively in the lower forms. It is proposed to call the intermediate cells (undeveloped forms) *astroblasts*.

The several different forms of glia cells, those possessing long processes, those provided with short processes (typical astrocytes), and still others in which the filaments come off from one or both poles only (brush cells), as well as the true ependyma cells, have all the same ultimate origin. Since Weigert's publication in 1890, and more especially since the appearance of his monograph on neuroglia, much attention has been given to the relation existing between the fibres and the cells in neuroglia.

The pictures given by the Golgi's silver stain seem to show a close union between processes and cell bodies, and to indicate that the former are mere protoplasmic elongations of the latter. The method of staining introduced by Weigert, and more or less modified by Mallory and Beneke, would seem to necessitate a modification of these views, since by its employment differentiation be-

tween fibres and cells in adult neuroglia has been rendered possible. According to Weigert, neuroglia, in human beings, consists of cells showing protoplasmic processes only during embryonic life; in the matured or adult condition, it is made up of a mixture of cells and fibres, in which the latter so greatly predominate that they are to be regarded as its chief constituent.

If we now turn our attention to the subject of tumors developing from neuroglia, we have no difficulty in referring certain well-known types to certain forms or stages of development of neuroglia. The spider-cell or Deiter-cell glioma, in which the cell processes are protoplasmic and still attached to the cell body, and those tumors in which the brush cells predominate, agree with astrocytes in the condition in which they occur during embryonic life; while such gliomata as are richer in fibres, which by the application of the new staining methods alluded to are separable into still further differentiated fibres and independent cells devoid of processes, may be taken to represent the adult neuroglia in human beings. It is further probable that a tumor corresponding with the intermediate cell, astroblast, which it is conceived may persist in human beings as a simple undifferentiated cell, may also arise. Such a tumor would have features in common with certain tumors now regarded as small-celled sarcomata.

Recently I have come into possession, through the kindness of Drs. W. W. Keen and H. M. Thomas of a brain tumor made up of cells which resemble, for the most part, the ependymal type of cells found in the human embryonic spinal cord, and present in certain animal forms throughout the whole lifetime of the species. These cells are arranged in a radial manner around blood vessels, toward which their protoplasmic prolongations are directed. The processes come together just before the vessel wall is reached, giving rise to a minute space between them at their points of junction and the walls of the vessels. I am of the opinion that these cells probably agree with

early ependymal cells, and that the tumor is to be regarded as a form of ependymal glioma. Although such a tumor has not yet certainly come to my notice, it is conceivable that the fully developed or adult ependymal cells may also give rise to tumors whose appearances would be different from the several forms already described.

The tumor formation in syringomyelia may not improbably be found to agree with one or the other of the types described. Indeed, in a case which I have seen recently the tumor mass was composed largely, if not exclusively, of cells of an early ependymal type.¹

Finally, the tumors of the retina, which agree in so many ways with the gliomata of the brain and cord, are capable of a similar classification. There is less that is remarkable in this statement when it is remembered that the retina is histologically as well as embryologically brain substance. Tumors consisting of astrocytes (spider cells) are known to develop from the retina; simpler and less highly developed gliomata, perhaps of astroblastic origin, are more common still; while tumors composed of cells which resemble, or are identified with, the cells of the layer of rods and cones (neural epithelium), of which I described the first specimen in 1891, have now become generally recognized. The layer of rods and cones agrees histogenetically with the epithelium lining the central canal of the spinal cord and the ventricles of the brain; it is, therefore, modified ependyma. If the rods and cones are regarded in the light of their embryological origin and histological relations, then the tumors composed of them are ependymal-celled gliomata; if, however, they are regarded in the light of the physiological functions which they perform, in which they behave as neural epithelium, then these tumors may properly be denominated neuro-epitheliomata.²

¹ This case occurred in the practice of Dr. Hudson, of La Fayette, Ala., who will soon publish it in detail.

² See Flexner, *The Johns Hopkins Hospital Bulletin*, 1891.

Clinical Cases.

DOUBLE OPHTHALMOPLEGIA CHRONICA EXTERNA.

Reported from the Clinic of Prof. M. Allen Starr, College of Physicians and Surgeons, New York. By Frederick Peterson, M. D., Chief of Clinic.

The following is the clinical history of a case of chronic external ophthalmoplegia, recently observed at the Vanderbilt Clinic, the notes having been taken by Dr. Goodhart and the writer:

W. A. G., male, born in 1872, single, came to the clinic complaining of weakness in his legs and inability to use his right hand in writing. He said that after walking a short distance his legs became exceedingly fatigued. His employment as a clerk had been interfered with of late because of tremor in his right hand, so that since August last he had had to give up writing altogether. He still keeps his position, and is a weighing-clerk in a coal office.

There is nothing in the family or personal history indicative of hereditary taint or predisposition to neuroses. He has never had syphilis. He has always been temperate, even abstemious, as regards alcohol, tobacco and venery. His birth was normal, though protracted. A younger brother, the only other child in the family, is living and well. In early childhood, before the age of five years, he had diphtheria, measles and whooping-cough, from which he recovered fully. His early mental and physical development was normal. He attended school until the age of 18 years, and received a good common school education. About the age of 5 years the patient received a blow in the neighborhood of the left ear, which left a small scar. At the age of 12 years he had what seems to have been an enlarged suppurating gland below the left ear, back of the ramus of the jaw. There was a discharge for some



FIG. I.

Case of ophthalmoplegia externa at age of 11 years, showing beginning of ocular palsies.



FIG. II.

Case of ophthalmoplegia externa at present, showing marked ptosis and over-action of occipito-frontalis. Facies expressive of mental dullness.

time, and a scar remains at the site indicated. There was a discharge about the same time from the left ear. He is completely deaf in the left ear, and the drum is absent. Shortly after this a slight drooping of the left eyelid was noticed, and later of the right. Gradually, from this time on, movements of the eyes became impaired, but neither the patient nor the family can give the order of affection of the various muscles. The patient asserts, however, that he never had diplopia, and that he had never noted any particular inconvenience in the use of his eyes, save from the drooping of the lids. The patient says he was stouter in proportion as a child than at present, and that he felt much stronger generally then than now. He dates a general feeling of weakness which he has from the onset of the ptosis.

The examination reveals a slimly built, rather poorly nourished physique. He is markedly stoop-shouldered, and the head is somewhat elevated in order to assist his vision. The face is rather immobile, and the expression of countenance suggests a mild degree of mental weakness. The most striking feature, as shown in the photograph, is the marked double ptosis. The eyes would be quite closed were it not for the strong over-action of the occipito-frontalis. All of the external muscles of the eye supplied by the 3d, 4th and 6th nerves are completely paralyzed, so that the picture presented is that of ophthalmoplegia chronica externa. The irides are not involved in the paralysis, and react normally to light and accommodation. The pupils are equal. A peculiarity noticed when a strong light is thrown upon the pupils is a rapidly alternating contraction and dilatation (hippus). Dr. Carter of the Eye department reports field of vision normal, myopia, and posterior staphyloma. Vision, R 15/40—1 D 15/30. L 15/100—1 D 15/40.

With the exceptions mentioned, all of the cranial nerves are normal. Sensation of all kinds is unimpaired over the whole body. The tendon reflexes of the upper extremities

are normal, perhaps somewhat hypertypical on the right side. The patellar reflexes are hypertypical, and there is slight ankle clonus on both sides. These reflexes in the lower extremities seem to vary from time to time in degree, being more pronounced at one time than another. The abdominal, cremasteric and plantar reflexes are normal. The sphincters are normal. In the upper extremities there is no marked muscular weakness, though with the dynamometer the left hand is stronger than the right (the patient is right-handed). To simple tests the flexor muscles of the thighs appear weak. The patient has noticed quick exhaustion and weakness in his legs after short walks for a year. Although typical Romberg symptom is absent, a peculiar rapid swaying, suggestive of tremor of the trunk muscles, is observed when the patient stands with his eyes closed. The usual tests reveal considerable ataxic or intention tremor in both hands, more marked in the right. There is a similar tremor of the neck muscles exhibited upon voluntary movement of the head from side to side or when bending forward to take a drink. The speech shows a slight hesitancy, which might be regarded as an approach to syllabic utterance, but I should not call it sufficiently typical to be designated as "scanning speech." The tremor of the right hand has interfered with his penmanship only since August, 1897. He has never had any pain in his head, trunk or extremities. He has never suffered from vertigo, nausea or vomiting.

I have seen photographs of the patient at various ages. The ptosis first showed itself in mild degree at the age of 12 years. Previous to this age the appearance of the face and eyes is normal.

Remarks.—Since von Graefe, in 1856, first described the syndrome known as ophthalmoplegia externa, over 300 cases have been reported in literature, many with autopsies. The pathological process underlying the disorder is not uniform. Peripheral lesions of the oculo-motor nerves, tumors at the roots of these nerves, and growths

or softenings in the quadrigeminal region may present a similar clinical picture. Furthermore, acute or chronic nuclear lesions are frequently at the basis of an ophthalmoplegia, and to this class of cases the term poli-encephalitis superior has been applied, because of the homology between some of the cases and cases of poliomyelitis. In acute lesions some ependymitis with punctate hemorrhages in the gray matter of the floor of the aqueduct of Sylvius has been found. The chronic form of ophthalmoplegia, to which the case just described belongs, is observed as an associated symptom in some of the chronic degenerative diseases of the nervous system, like tabes and general paresis. The chronic form is also met with in certain toxic dyscrasias (syphilis, diabetes, diphtheria, etc.). It is rather the rule, at least in the early stages, for the pupillary reactions to remain normal in chronic ophthalmoplegia dependent upon dyscrasias or focal lesions; whereas the internal muscles of the eye soon suffer in tabes and paresis. It is still doubtful if we have such a pathological entity as a true chronic degenerative process affecting only the nuclei of the various oculo-motor nerves, independently of any disorder elsewhere in the nervous system.

The case above reported presents certain interesting symptoms in addition to the ophthalmoplegia externa (intention tremor, increased reflexes, ankle clonus, slight hesitation in speech, and suggestive dulness of expression), which would make a multiple sclerosis more probable than any other cerebral disorder. It seems to the writer that sclerosis, with an unusual dissemination of the plaques, would explain all of the symptoms exhibited.

Critical Digest.

ON MULTIPLE SCLEROSIS, WITH ESPECIAL REFERENCE TO ITS CLINICAL SYMPTOMS, ITS ETIOLOGY AND PATHOLOGY.

BY B. SACHS, M.D.

Multiple or disseminated cerebro-spinal sclerosis has for many years been considered to be one of the most easily recognizable diseases of the central nervous system. Under the leadership of Cruveilhier¹ and of Charcot, the French school described the disease so accurately that there appeared to be little to add to the accounts as given by them. The typical forms are indeed unmistakable, but the disease appears so often under all sorts of disguises that its recognition may be by no means easy. Moreover, the interest in it is not exhausted by the establishment of the diagnosis, for it has important bearings to other diseases, and its etiology and pathology present many points for further elucidation. In America the disease is not as frequent as in France and in Germany; at all events, very few contributions have been made to the subject by American writers, and up to the present time I have found but one post-mortem record, and that an incomplete one (by Seguin), published in this country. The time seemed ripe, therefore, for a consideration of the disease; but the present writer will refer only to the more important questions that have been raised regarding it during the last few years, as the work of preceding years has been well summarized in the publications of Charcot, Marie, Gowers, Oppenheim, Dana, Pritchard, and Gray.

¹The bibliography (alphabetically arranged) will be found at the end of this article in the June number.

CLINICAL SYMPTOMS.

The following table of symptoms, as given by Charcot, may be taken as a starting point for the consideration of the symptomatology of multiple sclerosis. It need not be said that all the symptoms are rarely present, and that, according to the predominance of the several series of symptoms, the disease may be divided into a spinal, a cerebral, and a cerebro-spinal type.

I. SPINAL SYMPTOMS.

Positive.	{	Tremor on voluntary movements of the extremities—"Intention tremor" (arms and head; more rarely of legs).
		Titubation.
Negative.	{	Paresis (spasmodic) of the extremities.
		Contracture, with exaggeration of the reflexes—spastic rigidity.
	{	No sensory symptoms, or only very slight disturbance.
		Vesical disturbance, none or very slight.

II. CEREBRAL SYMPTOMS.

Dysarthria—slowness of speech; scanning of words.
 Nystagmus—blank expression.
 Attacks of vertigo—spasmodic myosis.
 Transitory amblyopia—white atrophy of the papillæ.
 Diplopia—associated paralysis of ocular muscles.
 Mental enfeeblement.
 Apoplectiform and epileptiform attacks.
 Difficulty in deglutition.

III. ABNORMAL OR UNUSUAL SYMPTOMS.

Trophic.	{	Muscular atrophies (amyotrophies), bedsores.
Tabetic.	{	Lightning pains.
		Romberg symptom.
		Anesthetic areas.
		Vesical and rectal paresis.
	{	Gastric crises.

Frequent remission of all the symptoms.

Charcot recognized three distinct periods in the development of the disease:

First period. The disease may begin with cerebral symptoms (attacks of vertigo, transitory diplopia, scan-

ning speech and nystagmus); more frequently the spinal symptoms are the first (a slowly progressive paresis to which intention tremor is soon added); in rare instances there is an apoplectic attack, preceded by vertigo and headaches, and followed by hemiplegia. The rarest of all onsets is the one with gastric crises, which are followed by other more characteristic symptoms. During the second period the typical symptoms are developed, and after years contractures set in. The third period is characterized by mental enfeeblement, speech becomes almost unintelligible, great physical weakness is developed, and finally there are vesical troubles, with subsequent cystitis, bed-sores, pyæmic conditions—death. Babinski claimed that an ascending acute myelitis was an occasional cause of death.

Marie has insisted upon a division into four different types, according to the mode of progression: 1. The chronic progressive type—a very gradual increase in all the symptoms and covering a period of years. 2. The chronic type, with exacerbation, marked by the occurrence of hemorrhages, sudden amblyopia, and the like. 3. The chronic remitting type—the progress is very slow, with only slight exacerbations from time to time. 4. The type characterized by permanent improvement and even cure. Almost all of the chronic disorders of the nervous system, tabes, for instance, could be subdivided in the same way. These divisions do not appear to be distinctive; and of the type leading to a complete cure we have seen no instance, and its existence is only a little short of conjectural. To Charcot we owe the recognition of imperfect forms of multiple sclerosis, in which the symptoms that have existed have disappeared. In these abortive forms (*formes frustes*) some of the important symptoms, as we shall see later on, are never developed, and in others the symptomatology is obscured by the occurrence of hemiplegic, tabic or of amyotrophic symptoms. Oppenheim has at various times directed special attention to the variability of the symp-

toms, the apoplectiform and vertiginous attacks, and to fundus lesions without functional defect.

The preceding statement is sufficient to show the extreme variability of the clinical pictures as a whole, and explains the possibilities of mistaken diagnosis. During the past few years some of the special symptoms enumerated in Charcot's table have received further elucidation. Struempell, who has a way of upsetting old medical beliefs, has maintained that the intention tremor of multiple sclerosis differs little, if at all, from ordinary ataxia. I have paid close attention to this point during the past year and have at present under observation three undoubted cases of multiple sclerosis, whose movements are distinctly ataxic. On the other hand, it cannot be denied that there are other patients suffering from multiple sclerosis, whose disturbance of motion is more in the nature of a tremor. We believe, too, that a typical tremor is often present at the beginning of the disease, and as the disease progresses the tremor becomes coarser, and then is scarcely to be distinguished from ataxic movements.

Struempell's proposition loses some of its apparent originality if we recall to mind the fact that Gowers in a foot-note (vol. ii., p. 548) states that *ataxic tremor* is the best translation for "Intentions-Zittern." To be sure, he chose the term Ataxic Tremor, "because there is no ataxia without voluntary movements." The difference between ataxia and tremor—a difference in degree only—can be brought out by asking the patient to write with chalk or lead pencil, when it will be found that the ataxic patient scarcely succeeds in the attempt to write, while the patient with tremor writes with some difficulty and in a jerky fashion.

The ocular symptoms of multiple sclerosis have not received the attention they deserve. The other morbid signs had been well understood long before these symptoms were properly described. Our knowledge of them was markedly advanced by the publication of Parinaud, who reported upon the findings in Charcot's cases, and by the

monograph of Uhthoff, who described with great detail the clinical and anatomical findings in the eyes of patients suffering from multiple sclerosis. Recent contributions to this special subject have been made by Luebbbers—a pupil of Uhthoff, by Guenther, Nagel, Kunn and Schwarz.

The commonest of the eye symptoms are the nystagmus and the nystagmus-like movements. These are most apt to appear on extreme lateral movements of the eyes, and are generally associated in character. While occasional twitching movements of the ocular muscles may occur in healthy persons, regularly developed oscillatory movements upon each lateral excursion are characteristic of the disease. Of course, it should be borne in mind that the symptom is present in other diseases of the nervous system, of which mention will be made later on.

Kunn described a condition which he calls a “fixation tremor” setting in when the eyes are moved from vision straight ahead to the fixation of a definite object. The same author calls attention to a concomitant strabismus which he interprets to be a dissociation of ocular movements that were previously associated.

Luebbbers mentions a case in which the nystagmus appeared with converging movements of the eye; but this is entirely exceptional. The nystagmus may be horizontal, vertical, diagonal or rotary. Raehlman and Schwarz dwell upon the special form of ataxic nystagmus, which is practically an uncertainty of movement, the eyes simply overleaping the mark in passing from one point of fixation to another. The same phenomenon is observed in Friedrich's disease, and can be distinguished from the more rapid movements of typical nystagmus. True nystagmus occurs in a majority of cases of multiple sclerosis (according to Uhthoff, in 58 per cent., and according to Marie, in 70 per cent. of all the cases). The variations in the percentages are accounted for by the fact that Uhthoff draws a distinction between nystagmus and nystagmus-like movements. These oscillatory movements of the eyes constitute, therefore, one of the most constant and reliable

signs of multiple sclerosis. In view of their frequency, however, it is well to remember that they occur with different forms of congenital defective development (idiocy, hereditary spastic paraplegia, etc.), also in Friedreich's disease, in hereditary cerebellar ataxia, and in a few cases of syringomyelia. Its occurrence in these various diseases is responsible for much of the confusion that has arisen in the attempt to differentiate between them.

Uhthoff is responsible for the statement that affections of the optic nerve are frequently associated with true nystagmus in multiple sclerosis, whereas we are more apt to have nystagmus alone without such atrophy in other conditions.

The actual cause of nystagmus has not yet been fathomed. Uhthoff thought it was due to lesions in the brain, and particularly in the medulla oblongata; but he also concedes that it might be due to changes in the peripheral nerves supplying the ocular muscles. A lesion involving the posterior longitudinal fasciculus was supposed by some to explain the occurrence of nystagmus; but, as Redlich has pointed out, this tract has been entirely exempt in cases of multiple sclerosis with marked nystagmus, and in some cases in which nystagmus was never present, this same bundle was found to be seriously affected. In all probability, any lesion interfering, by lesions of the nuclei or of the associating tracts, with the regular movements of the ocular muscles, would be sufficient to account for nystagmus.

Paralysis of the ocular muscles is not an infrequent accompaniment of multiple sclerosis. It has been present in three of ten cases which have come under my observation within the past two years. The abducens is most commonly affected. Next in order of frequency comes the oculo-motor nerve, which is, however, rarely affected in its entirety. Occasionally the trochlear (IV.) nerve is involved. A point of some importance is that these palsies are variable, disappearing in some weeks or

months, and occasionally returning again to as marked a degree as before. The remission in this one symptom is as characteristic of multiple sclerosis as the remission in most of the other symptoms, and it is the occurrence of such remissions that makes it particularly difficult to distinguish between disseminated sclerosis and multiple cerebro-spinal syphilis (Oppenheim, Sachs).

In addition to single ocular palsies, there may also be paresis or paralysis of associated movements. The upward and downward movements are, as a rule, not affected (Parinaud, Marie, Uhthoff, and Luebbers). Parinaud claimed that paralysis of convergence was frequent, but Uhthoff found this condition in only three per cent. of his cases. A complete ophthalmoplegia externa has not been developed in multiple sclerosis. The direct cause of ocular palsies is to be found in the sclerotic patches occupying the region of the various ocular nuclei. Positive and anatomical proof of this would still be welcome.

By way of contrast with the early appearance of ocular palsies in *tabes dorsalis*, it may be noted that in multiple sclerosis the palsies do not appear, as a rule, until long after unmistakable symptoms of the disease have appeared. The pupils may be unequal, and sometimes react sluggishly to light and during accommodation, but iridoplegia is very rare. Charcot insisted upon the great value of this preserved light reflex as a factor in differential diagnosis. Damtsch claimed that the condition of *hippus*—a constant changing from contraction to dilatation of the pupil—is often found in multiple sclerosis. He likens the *hippus* to the increase of the tendon reflexes, but the analogy is not a very happy one.

Much more characteristic of multiple sclerosis than any and all of the preceding ocular symptoms are the changes in the visual power, in the visual fields, and in the structure of the papillæ and the optic nerves. The subjective disturbances are entirely out of proportion (i. e., relatively less), to the objective changes. Complete blind-

ness is the rarest of all occurrences, although transitory blindness has been observed (Schwarz). Complete recovery from the visual defect of multiple sclerosis is possible. According to Uhthoff, Luebbers and others, central scotomata are most frequent. In this area the color sense is the one most disturbed—generally for red and green, sometimes for all colors. These central anesthetic areas may occur together with the normal peripheral fields, or with a mere diminution of peripheral vision. In some cases there may be a peripheral contraction of the visual field with normal central vision. Concentric limitation of the fields has been described by Oppenheim, Thomsen, Buzard and Uhthoff, but only in cases in which an hysterical or a functional condition was superimposed upon disseminated sclerosis. Almost every writer who has investigated this special subject refers to the disproportion existing between the visual defect and the ophthalmoscopic appearances. In one of Luebbers' cases the visual defect was in keeping with the changes in the papilla, but in two others there was only a very slight atrophy of the papilla, and yet vision was much diminished and the visual fields were distinctly affected. In still another instance there was a marked functional disturbance without ophthalmoscopic change. A few other authors have described distinct changes in the papilæ without any functional disturbance. The visual symptoms generally appear long after the others have been developed, but may come on with extreme suddenness (Uhthoff and Buss).

Gowers and Marie have called attention to the fact that the visual disturbance of multiple sclerosis may be unilateral.

Ophthalmoscopic examination has revealed several peculiar changes. Complete atrophy of the optic nerves is very rare. Uhthoff found it in only 3 of 100 cases. The rarity of this is worthy of note, and is probably due to the frequent preservation of the axis cylinders, in spite of the destruction of the medullary sheath. Incomplete atrophy

—discoloration of the entire papilla—the peripheral parts being more distinctly altered than the nasal half, occurs in about 50 per cent. of all cases. Atrophy of the temporal halves of the papillæ is almost pathognomonic of multiple sclerosis. Charcot insisted that the papilla retained a yellowish color in contradistinction to the white appearance in the atrophy of tabes. Optic neuritis has been found in a number of cases (in three of Luebbers' cases). Partial temporary atrophy is, according to Uhthoff, a common symptom in intoxication of amblyopias, tobacco and alcohol); but in these the atrophy is always bilateral, whereas it is frequently unilateral in multiple sclerosis. This special resemblance to the intoxication amblyopias is of interest, if we remember that multiple sclerosis has been shown to be at times of toxic origin.

The anatomical examination of the eyes (Uhthoff, Luebbers, and others) has revealed conditions as variable as are the functional disturbances. It has been shown that there are widespread atrophic changes in the optic nerves, depending probably upon the proliferating process emanating from the connective tissue, and gradually involving the nerve fibres; the sheaths are often destroyed, but the axis cylinder is, as a rule, not affected; in other words, the process is interstitial in origin, the changes in the nerves being secondary. In consequence of this preservation of the axis cylinders, the secondary degeneration does not ensue, and the nerve fibres do not forfeit their functional power.

It is very evident, as will be shown in the section on pathology, that the sclerotic changes in the optic nerves are very much the same as in the other parts of the central nervous system. The next question is, whether or not the changes in the connective tissue are due to the disease of the blood-vessels. No unanimity of opinion has as yet been reached on this point, and it is more than probable that the process begins differently, according to the varying etiology of the cases. Uhthoff, Buss, Taylor and others

have paid especial attention to the behavior of the blood-vessels. Several of these writers have found an increase in the number of the finer blood-vessels, and a change in their walls, followed by a proliferating process in the surrounding tissue.

Nolda, who studied the relations of multiple sclerosis to infectious diseases, thinks that the sclerotic patches take their origin from the walls in the blood-vessels. Luebers, in comparing the changes in multiple sclerosis with those found in the primary optic atrophy of tabes dorsalis, finds that in multiple sclerosis there is more of a focal degeneration than in tabes, and, above all, there is in tabes no such number of normal axis cylinders as in multiple sclerosis. The optic atrophy of tabes is due to the primary degeneration of the optic nerve fibres, while in multiple sclerosis the atrophy is the result of an interstitial neuritis, and this in turn may be due to a disease in the blood-vessels. In multiple sclerosis, it may be added, the retinal elements are not affected.

Scanning speech has long been considered a characteristic symptom of multiple sclerosis; but it would be a mistake to suppose that this is the only speech disturbance common to this disease. Putting it broadly, almost every form of dysarthria is possible in multiple sclerosis, for the disturbance is due to lesions in the pons or medulla interfering with the speech mechanism. Leube found a disturbance of innervation of the vocal cords in one instance, and I have recently seen a typical case of the disease in which the opposition of the vocal cords was so imperfect that the patient spoke in whispers. In other cases nasal speech has been noted, and in still others true bulbar speech has been reported. In the writer's own experience, there is often an evident speech tremor not unlike that observed in the early stages of general paresis, and reminding one somewhat, too, of the peculiarities of speech in some patients suffering from Friedreich's disease. Disturbances of articulation, if excessive, are apt to be associated with

other symptoms pointing to an involvement of the various nerve nuclei in the pons and the medulla oblongata (cases of Joffroy, Leyden, Berlin, Fuerstner, Redlich and others). If these symptoms predominate, they constitute the special bulbar form of multiple sclerosis. This type was carefully considered by Hallopeau.

An interesting example of this class of cases was recently reported with autopsy by Schuster and Bielchowsky. In this instance nasal speech was associated with paresis of the left arm and the left leg, with dyspnœa, headaches and double vision. There was also divergent strabismus of the right eye, as well as paralysis of both sixth nerves. The pupils and fundi were normal. A focal lesion in the pons was suspected, but the autopsy revealed multiple sclerotic foci in the pons and medulla and in other parts of the brain.

Aside from the peculiarities in the development of single symptoms, abnormalities occur in the development of the entire clinical picture of multiple sclerosis. In the literature of the subject the *formes frustes*—the aborted types of multiple sclerosis—have given rise to considerable discussion. It is well to bear in mind that there is no regularity in the development of sclerotic patches, whence it follows that departures from the typical forms of the disease will occur readily enough. Slightly irregular forms do not, therefore, deserve the designation "*formes frustes*."

Charcot was the first to dilate upon these special types, referring particularly, 1. to cases in which certain symptoms, which had existed, had disappeared; 2. to cases of which the symptomatology had never been complete—such cardinal symptoms as the tremor or the nystagmus never having developed; 3. to cases in which the type has been altered by the intervention of other symptoms of an hemiplegic, tabic or amyotrophic order.

The establishment of the *formes frustes* has opened the door to diagnostic errors; for a multiple sclerosis may ap-

pear under the guise of an hemiplegia, of a transverse myelitis, or of an amyotrophic lateral sclerosis. In the opinion of the present writer, the diagnosis of multiple sclerosis should be made only if the cardinal symptoms of the disease, such as nystagmus, scanning speech, intention tremor are present in addition to the other symptoms of myelitis, hemiplegia, etc. The variability of the symptoms (Oppenheim) argues in favor of multiple sclerosis, as do also the recurrent apoplectic and vertiginous seizures. Oppenheim has laid especial stress upon the occurrence in such cases of the characteristic changes in the fundus, with little or no functional impairment of vision.

Apoplectic attacks may occur in the course of a multiple sclerosis, but occasionally, as in a case now under my observation, hemiplegia is developed slowly at the outset of the disease, and is then scarcely to be differentiated from other forms of apoplexy; excepting that in these cases which affect men and women in middle life the usual etiological factors of an apoplectic seizure, cardiac disease, or specific endarteritis are generally absent. If the person who has suffered from an apoplectic attack develops marked nystagmus, scanning speech or ataxic movement in the paralysis of the upper extremity, the suspicion of multiple sclerosis may well be entertained. Cases of this character have been described by Werner, Bikeles and others. The question arises whether these apoplectic attacks in multiple sclerosis are due to vascular disturbances or to a peculiar localization of the sclerotic patches. I am inclined to side with Charcot, Redlich and Babinski, who believe that these forms are due to the early development of sclerotic patches in the pons and medulla oblongata, although Taylor has shown that such patches also occur in the cortex and other parts of the brain. Witzel has published a case in which there was a crossed paralysis of the facial and abducens nerve of the opposite side.

Many writers have conceded the occurrence of acute and chronic cases of multiple sclerosis, presenting none

but spinal symptoms (Bourneville, Guérard, Leyden and Oppenheim have recorded cases of acute, transverse myelitis which were eventually proven to be cases of multiple sclerosis). In the absence of the ordinary etiological factors (transverse myelitis trauma, syphilis, vertebral disease) the suspicion of disseminated sclerosis may be entertained, but the cardinal symptoms of the disease must be in evidence to prove the suspicion.

The purely spinal and chronic forms may appear under the clinical guise of a spastic spinal paralysis. For months, and even years, no other symptoms may be forthcoming. No doubt, some of the cases of primary lateral sclerosis were in reality early forms of multiple sclerosis. Cases of this description have been reported by Krueger, Ormerod, Lapinski and Redlich. A very typical case of this character was observed recently in my department at the Polyclinic, to which it had been referred by Dr. A. Wiener. In all such cases the occurrence of the characteristic changes in the fundus will lend valuable support to the diagnosis of multiple sclerosis. However clearly the symptoms may be developed, and however carefully, multiple sclerosis may be confounded with other diseases, above all with cerebral spinal syphilis (Oppenheim, Sachs and others). The remissions in the symptoms, the preponderance of the spasticity over the paralysis, the apoplectic seizures may be characteristic of both diseases. Greater stress, in my opinion, should be laid upon the pupillary changes in syphilitic affections (absolute immobility, unilateral pupillary symptoms and irregular contour of the pupils), and the presence in multiple sclerosis of nystagmus, intention tremor and scanning speech, which are, after all, extremely rare in syphilis of the brain and of the spinal cord.

There will be, as a rule, no difficulty in differentiating between multiple sclerosis and paralysis agitans; but Schultze and Sachs have reported upon cases in which the symptoms of multiple sclerosis have been associated with those of paralysis agitans; in one young patient of

the latter writer, who exhibits most of the symptoms of paralysis agitans, there are also a nystagmus and marked nasal speech. In another patient, whose condition was at first diagnosticated (by competent observers) as functional tremor, then as intention tremor of multiple sclerosis, later developments have shown the disease to be a typical form of paralysis agitans. In view of such experiences, we may maintain that the clinical distinction between multiple sclerosis and paralysis agitans is not as absolute as it has been supposed to be since the days of Charcot.

We may concede that hysteria may simulate multiple sclerosis. More often hysterical symptoms are present in addition to those of multiple sclerosis. The differentiation will depend largely upon the presence of such distinctly hysterical stigmata as are foreign to the pure type of multiple sclerosis. Westphal's two cases, in which the anatomical changes were wanting, although the patients exhibited all the symptoms of the disease, have led to much discussion.

French writers have called them hysterical without much hesitation, and Struempell has chosen to establish a separate form of pseudo-sclerosis, bringing under this head what appears to us a rather heterogeneous number of cases. He has allied with this form a diffuse sclerosis of children; but the clinical types which he attempts to establish need careful examination before they can be adopted.

This leads to the consideration of multiple sclerosis in early life. The differential diagnosis between it and other diseases of infancy presents so many difficulties, that much care should be exercised in making the diagnosis, or in accepting it when offered by others. In a recent article by Struempell, in which the author challenges criticism on other points, he insists that the diagnosis of multiple sclerosis in children is not certain, unless corroborated by post-mortem findings, and to this conservative view we are inclined to subscribe.

The first case of multiple sclerosis in a child appears to have been described by Schuele (1871). This was followed by reports of Dreschfeld, Bristowe and Ten Cate Hoedemaker. Marie, in 1883, reported upon cases of his own, and collected 13 in all. In 1887 Unger had increased this list to 19. Six years later, Totzke, reporting 2 cases of his own, tabulated 39 in all. One case was reported by the present writer in his book on the "Nervous Diseases of Children," which Stieglitz has, through a printer's error, called a doubtful case in the latest publication on this subject. Stieglitz cites three cases of his own, and tabulates 35, evidently excluding some which he considered doubtful. In addition to those diseases of the adult which enter into the discussion of the differential diagnosis, such as syphilis, transverse myelitis, hysteria, we must especially consider the infantile spastic palsies, hereditary spinal ataxy (Friedreich's disease), hereditary cerebellar ataxy and hereditary spastic paralysis—the last has been omitted from Stieglitz's carefully prepared list.

As for the infantile cerebral palsies, the ordinary paraplegias (birth cases and cases of defective development) will generally be ruled out by the history of early onset, by the frequent occurrence of convulsions and by defective development, which is more marked than in cases of multiple sclerosis.

The acute hemiplegias, with their characteristic symptoms, cannot give rise to any confusion. Some doubt could be entertained, however, with respect to the cerebral diplegias. Nystagmus is common enough in these; intention tremor, or, rather, ataxic movements, are observed frequently, and disturbances of speech (bradylalia) are very common. It is very rare, on the other hand, to find any two of these symptoms in a case of diplegia. Furthermore, it should be remembered that a cerebral diplegia of an acute onset is a rare occurrence, and rarest of all in patients beyond the age of five to seven years. These cerebral diplegias present extreme contractures and

post-paralytic disturbances of motion (athetosis and the like), which seldom occur in multiple sclerosis. As a matter of fact, of the large number of cases of cerebral diplegia which the present writer has seen, there has not in a single instance been reason to suspect the presence of multiple sclerosis. In making the diagnosis, the entire congeries of the symptoms should be considered, and not any one clinical sign. Stieglitz says, correctly, that the question is whether infantile cerebral palsy has been excluded in cases diagnosticated as multiple sclerosis.

From Friedreich's disease differentiation is generally easily made, although every now and then patients come under one's observation whose clinical symptoms may leave some doubt as to the presence of one or the other of these diseases. A resemblance may be due to the peculiarities of speech, to the awkward movements of the upper extremities in Friedreich's disease, which may simulate an intention tremor, and to the presence of nystagmus; but the condition of the reflexes in disseminated sclerosis, the optic atrophy and the absence of true ataxia of the lower extremities will help to establish the differential diagnosis; and, after all, in Friedreich's disease the disturbances of speech are slow and labored, but rarely of the typical scanning order.

At a recent meeting of the Neurological Society I presented an adult patient in whom the symptoms pointed to Friedreich's ataxia, and yet there was a bare possibility of his having a bulbar form of multiple sclerosis. This patient was 39 years of age, his past history was good, without any evidence of preceding syphilis. At the age of 20 he was weak in the knees, and frequently made missteps. In 1887 he had sought medical advice because of difficulty of locomotion, noticed especially in climbing stairs. When examined in March, 1895, he complained chiefly, while walking, of weakness in the extremities and slight difficulty in speech. He exhibited an ataxic spastic gait and static ataxia. The pupils reacted to light and

during accommodation. The patellar reflexes were absent. There was distinct ataxia of the right upper extremity. He had a form of speech which was midway between a slow and a bulbar speech. There was slight atrophy of the optic nerves. The case was at first supposed to be one of locomotor ataxia, but the further development of the disease showed that it was either Friedreich's ataxia or a bulbar form of multiple sclerosis. The intention tremor, the optic atrophy and the peculiarities of speech would be in harmony with the diagnosis of multiple sclerosis; but the absence of the knee-jerks, ataxia of the upper extremities, which was not a typical intention tremor, and the normal behavior of the pupillary reflex were in keeping with Friedreich's ataxia, which appears, however, as a rule, early in life. The first symptoms in this patient's case appeared at least as early as the age of 20 years, and possibly earlier.

Still greater difficulty, however, is experienced in the attempt to differentiate multiple sclerosis from hereditary cerebellar ataxia (type, Nonne-Marie). This is well illustrated by Stieglitz's Case II., in which the diagnosis of multiple sclerosis hangs by a hair, and the author himself does not appear to be firmly convinced of the diagnosis. If a previous history of ptosis, of a paresis of the internal rectus muscle, and the Romberg symptom do not support the diagnosis of hereditary cerebellar ataxia, it is also certain that swaying of the body, with the eyes open and closed, is not very usual in multiple sclerosis. In a case like this, as well as in the one cited above by me, the post-mortem examination alone will satisfactorily determine the nature of the disease. It is well to insist again upon the fact that the mode of onset, the time of life at which the disease is developed, as well as the history of some preceding illness, will often help us to differentiate between the two diseases.

Hereditary, spastic paralysis, as described by Newmark, Pelizaeus and others, should be considered in this con-

nection. There is a striking resemblance in the symptoms, but nystagmus, intention tremor and scanning speech are not typical of the hereditary form of spastic paralysis. Only a few months ago a boy, 12 years of age, came under my observation, who, after a fright, had developed marked spastic paralysis of the lower extremities, with slight ataxic movements of the right upper extremity, and a peculiarity of speech, which was slow and deliberate, rather than scanning. There were no nystagmus, no pupillary abnormalities, no optic atrophy—in short, no symptoms that were at all characteristic of multiple sclerosis, except the speech. In this case the fact that the boy was absolutely well until the age of 12, that there was no history of any similar occurrence in any member of his family, and that the disease came on in rather abrupt fashion, argued largely in favor of multiple sclerosis; and yet, but for the slight ataxic movements of the right upper extremity and the peculiarities of speech, there was little to throw the balance in favor of disseminated sclerosis. In cases of this description time is needed for the development of further symptoms, which will enable one to establish a diagnosis with some degree of certainty.

(To be concluded in the June number.)

Society Reports.

NEW YORK NEUROLOGICAL SOCIETY.

Stated Meeting, April 5th, 1898.

B. Sachs, M.D., President.

REPORT OF TWO CASES OF BRAIN TUMOR.

Dr. Alfred Wiener read this report. In the patient presented, the tumor was revealed by operation; in the other the specimen was obtained by autopsy.

Case I. A male, twenty years of age, was brought to his clinic in June, 1897. He was born of apparently healthy parents, but the father was alcoholic. He was a seven-months baby, but was born apparently in a normal manner. He was slow in learning to walk and talk. When about seven years old, he had otitis media, followed by a profuse otorrhœa. The present trouble first announced itself while he was playing cards; suddenly the cards dropped from his hands, and his face "felt stiff." He insisted that he did not lose consciousness, and that he felt as usual by the next day. Previous to this he had suffered from frequent and severe headaches, associated with vomiting. The pain was usually frontal. One month after the attack just described, there was a second and more severe attack. He remained dazed for about two hours; speech was slow, and he could express his thoughts only with difficulty. He slowly became weaker, and vision failed. When first examined, there was no perception of light in the right eye; with the left eye he could see fingers at four feet; the pupils reacted to light; the veins were engorged; the disk was swollen and hazy. The tongue was protruded straight. The dynamometer showed marked loss of power on the right side. The patellar reflexes were absent on both sides. There was motor aphasia. Physical examination showed the heart, lungs, kidneys and spleen to be normal, and the blood examination revealed a leucocytosis. There was

increased excitability on the right side to the electric current. The boy appeared fairly well nourished, but was distinctly apathetic. Smell, and taste, and muscular sense were unimpaired. These symptoms pointed to a tumor. Abscess was excluded for the reason that there was no source of infection. The history was negative regarding tubercular and syphilitic infection. He had been unwilling at first to venture upon the diagnosis of the location of the neoplasm. The possibility of multiple tumors was borne in mind. After four weeks the reflexes in the lower extremities began to return. The headaches became more severe and the disturbance of speech more positively that of motor aphasia. He had, therefore, come to the conclusion that the tumor was a cortical one, involving the face and arm centres, and the posterior extremity of the third frontal convolution—i. e., the speech centre. During this time the man had been on anti-syphilitic treatment, but with negative results. The slow growth of the neoplasm led him to believe that it was a glioma. An operation was advised because he was reasonably sure of the location of the growth, and because a trephine operation seemed justifiable even if the tumor could not be removed. The patient was operated upon by Dr. A. G. Gerster on July 31, but the operation through the scalp and skull was attended by such great loss of blood that further interference was postponed. A few days later the operation was resumed by Dr. H. Lilienthal. A piece of the tumor was removed, and proved on microscopical examination to be a telangeiectatic glioma. Any attempt at manipulating it resulted in severe hemorrhage. Some days after the operation a small abscess was found near the surface, and this was incised and evacuated. After this improvement was more manifest. On October 12th it was noted that the patient was irritable and, at times, quite excited. He showed some ataxia in his movements; the speech was distinctly hesitating; the patellar reflexes were present, but much diminished. His muscular sense was still present. On November 1st another localized abscess was detected and evacuated. The tumor had been steadily growing out through the wound. On January 18th it was noted that the patient could no longer express the names of familiar objects. On January 28th the mass measured $4\frac{3}{4}$ inches in length, $2\frac{3}{4}$ inches in width at the

upper portion, and $3\frac{1}{4}$ inches at the lower part. On February 8th, when he was just recovering from an attack of influenza, it was found that he could no longer walk alone, and that he was much slower in comprehension than formerly. On February 23d the tumor measured 5 inches in length, 3 inches in width at the upper portion, and $3\frac{1}{4}$ inches at the lower portion. During the month of March he became more apathetic, and could not repeat his own name. At the present time, there is paresis of the left facial and right upper and lower extremities; the patellar reflex is present on both sides; there is double abducens palsy, more marked in the left eye; there is still a tendency to fall toward the right side and backward. The muscular sense on the right side is very much affected. The tumor now measures 5 inches in length, $3\frac{1}{4}$ inches in width at the upper part, and $3\frac{1}{2}$ inches at the lower part. There have been no severe headaches or epileptic seizures. It is probable that the tumor has been growing just as extensively within the skull as on the outside. The forced laughing, the double abducens palsy and the aphasia all point to extensive involvement of the cortex by the growth.

Case II. A boy of seven years, seen first on October 19th, 1897. The child was born of healthy parents, but the father was alcoholic. The patient was the second child, and was born after a normal labor at full term. He was perfectly well up to twenty months old, at which time he apparently had an attack of rheumatism, lasting five weeks. From then up to a short time before coming under observation he was well developed, both physically and mentally. It was then noticed that he went down stairs clumsily. About this time he came home one day with a marked swelling over the right frontal and parietal region, and said that a man had struck with his fist over this part of the head. After this he suffered from severe headaches, and about the same time it was noticed that he was not as bright as formerly. It was next noticed that fluids would run out of the mouth while he was drinking. On October 19th, examination revealed a paresis of the two lower branches of the right facial nerve, without any electrical change. The tongue, when protruded, deviated toward the right. There were analgesia and slight dullness to touch on the right side of the face. The

patellar reflexes were absent on both sides. There was no bladder or rectal trouble, and there had been no epileptic seizures. Although appearing distinctly apathetic, he was fairly intelligent. There was also a double optic neuritis. No evidence of syphilis or tuberculosis could be found. Abscess was excluded for the same reason as in the first case. On account of the complete hemi-paresis and hemi-anesthesia, without loss of the muscular sense, the tumor was thought to be subcortical, and located near the internal capsule. The tumor was suspected to be a glioma on account of location and the symptoms. He was put upon antisyphilitic treatment. On October 23d the patellar reflexes were found to be present, but very much diminished, and anesthesia was more prominent, especially in the right upper extremity. On November 11th the optic disks on both sides showed marked atrophy, and small hemorrhages could be seen in the retina. He walked with a distinct hemiplegic gait. On November 16th, anesthesia was quite marked in the hand and face, and the right arm was totally paralyzed. Up to December 2d the condition remained about the same. The patellar reflexes were exaggerated on both sides. On account of the extensive paralysis of the arm and face, and slight involvement of the leg, he had been inclined to believe that the greater part of the tumor occupied the centrum ovale. The antisyphilitic treatment had been given a fair trial without the slightest improvement. On January 20th, having recovered meanwhile from an attack of scarlet fever, it was found that there was spasticity on the paralyzed side, that he was unable to walk and was totally blind. On February 7th he died, having been intelligent to the last moment. The autopsy was made by Dr. Wiener four hours later. The abdominal and thoracic organs were normal. The brain weighed 52 ounces. The convolutions over the left hemisphere were very much flattened. The left hemisphere was decidedly softened, and horizontal sections through it revealed a soft, infiltrating tumor occupying the entire centrum ovale. In the lower portion of the hemisphere the tumor was mainly frontal. The microscopical examination showed it to be a gliosarcoma.

Dr. Wiener said that the absence of the patellar reflexes had been reported by others, but no satisfactory explanation

had been offered for this phenomenon up to the present time. Frontal ataxia, which was really due to involvement of the trunk muscles, which have their location in this particular portion of the brain, was another interesting point. It was worthy of note that in both cases the father was alcoholic. In the second case, as there was one symptom present before the trauma, he was inclined to believe that the trauma only served to make the pathological condition manifest. The relief afforded by the trephining was evident in the first case.

Dr. C. L. Dana said that in the specimen he noticed a portion of the tumor lying back of the Rolandic fissure. This, he thought, might explain the ataxia. He had recently seen a large abscess of the frontal lobe in which there had been no history of ataxia. It seemed to him straining the point to try and make it a frontal ataxia. It could not be from the involvement of the trunk muscles.

Dr. Arthur Booth said that in the three cases of frontal tumor that he had seen, there had been no ataxia, and the reflexes had been absent after the symptoms had been well developed.

Dr. Joseph Fraenkel said that he was convinced that the condition of the knee-jerks was entirely dependent upon the contraction of the muscles. Another factor, of course, is the muscular sense, and still another is the difficulty of determining clinically slight disturbances of deep sensibility. In the case under discussion he would explain it by invasion of the frontal and parietal lobes, and the probable more or less marked disturbance of the deep sensibility.

Dr. W. M. Leszynsky said that the more he tested the knee-jerks, the more he thought the theory of inhibition could be, to a great extent, sustained. This had been particularly noticeable in cases of meningitis in children that he had observed in the early stages. These children had been previously tested for their knee-jerks as a matter of routine. When the signs of pressure had set in later, the knee-jerks had become exaggerated, and this he attributed to the irritation within the brain. A similar explanation could be offered in connection with the case now under discussion.

Dr. B. Sachs thought the question of ataxia in these cases could not be settled by the supposition that it was wholly due to the invasion of the tracts lying in the vicinity of the parietal lobule, for the reason that ataxia had been noted in other cases of frontal tumor in which there had been no involvement of that particular region. It seemed to him, therefore, that the ataxia had come to be looked upon as a symptom of frontal tumor. The behavior of the reflexes in the cases reported seemed to him very remarkable. In his own mind the older theories were of greater help than the theory suggested by Van

Gehuchten, or that propounded by Fraenkel. He believed Dr. Fraenkel was confounding things which are coincident, without necessarily being related as cause and effect. It would not do simply to assume that there were changes in the deep sensibility which we were as yet unable to detect clinically. He was inclined to believe that the same causes which produce loss of reflex are also responsible for the changes in the tonus and in the deep sensibility. There was probably some additional fact in the explanation of the reflexes which was yet lacking, but we should not endeavor to make the facts fit the theory. Another interesting point in this case was the certainty with which it was diagnosticated as a subcortical tumor. The hope of an excision had been abandoned at a very early day. The diagnosis of its situation had been largely based upon the non-occurrence of epileptiform seizures from the beginning to the end of this disease.

Dr. Wiener, in closing, said in reply to Dr. Dana, that he did not doubt that there might be tumors of the frontal lobe without any ataxia, but it had been shown when the portions bordering on the frontal convolutions were destroyed, there was this peculiar cerebellar gait. If Dr. Fraenkel's theory regarding the reflexes were correct, he could not understand why, in the case just reported, the reflexes should have returned afterward. He thought the increased reflexes were due to the destruction of the normal inhibitory influence.

A CASE OF SUBCORTICAL VISUAL APHASIA.

Dr. Joseph Collins reported such a case, and presented the brain. The patient, a male, fifty-eight years of age, had not had syphilis, rheumatism or gout; nevertheless the vessels showed well-marked arterial sclerosis. For five months he had been complaining of headache and vertigo. Then, without warning, he became unconscious, and lay in a stuporous condition for about three weeks. After this he recovered his mental balance, but was unable to return to work because, it was said, of his "weakness." Examination of the eyes showed complete right lateral homonymous hemianopsia. There was complete word blindness, and almost complete letter blindness. He could not mark a word off into syllables. There was no object blindness. He recognized things and called them by their right names. He wrote very badly to dictation, and this showed that he could not retain the words in mind long enough. When asked to read what he had written, he pointed to each word and said: "I would like very much to read," and continued to repeat this again

and again. He did not give the slightest heed to words put in writing, although he responded promptly to spoken commands. Spontaneous speech was not materially disordered. The symptoms pointed to a lesion between the angular gyrus and the visual centres, situated in the posterior end of the left hemisphere. If the lesion were in the primary visual centre, there would be object blindness.

The patient had been admitted to the City Hospital, and extended observation showed no material change in the above findings. He was easily excited. On November 8th, 1897, he suddenly fell over in a general convulsion. He did not recognize any one after that, and soon developed a right-sided hemiplegia. The autopsy was made a few hours after death. There was no noticeable abnormality, aside from the brain, except an extensive arterial sclerosis. The entire occipital lobe was reddish-yellow, and, with the exception of the extreme posterior cortex, the brain was replaced by a cystic formation. Nearly all of the lingual gyrus and the cuneus had been destroyed—in short, the cystic formation involved the whole posterior part of the left hemisphere save the very apex of the cuneus, and particularly the inner surface. The remainder of the left hemisphere was apparently normal. The temporal lobes were not implicated. The depth of the lesion was almost through the thickness of the white matter and into the ventricles. Both lateral ventricles were distended with clots.

PHILADELPHIA NEUROLOGICAL SOCIETY.

February 28th, 1898.

The President, Dr. F. X. Dercum, in the chair.

Dr. Simon Flexner, of Johns Hopkins University, read a paper on

GLIA AND GLIOMATOSIS. (SEE P. 306).

Dr. Joseph Sailer said that Dr. Flexner's paper had brought before us some novel ideas in regard to the histology and development of gliomata. He wished to know whether Dr. Flexner, in the study of these various forms of gliosis, had not observed cells that in some respects were atypical, and which it would hardly be fair to regard as ependymal in their origin. He had recently found cells showing a slight projection on one side in the cortical region of the brain. These were large, pale, and contained a large faintly-stained nucleus. This tissue could not be hardened and stained so as to show the relation of the cells to the neuroglia fibres. Deeper down he had found peculiar cells, pyramidal in shape and quite irregular in type. Both forms were situated in hyperplastic neuroglia tissue, composed of bundles of fibres arranged irregularly with large spaces between them. He thought that these resembled the cells described as ependymal.

Dr. Spiller stated that the undifferentiated glia cells of v. Lenhossék, which are supposed to give rise to the cells of the gliomata, are purely hypothetical, and that since we know that glia cells and fibres are separate from one another, these hypothetical cells are not needed to enable us to understand the proliferation of the glia. He referred to the recent excellent papers by Rossolimo, Henneberg and E. W. Taylor on the subjects of glia and gliomatosis, and spoke of the gliosis which is so commonly seen about the central canal in spinal cords which do not seem to be abnormal in other respects. This gliosis may have some bearing on syringomyelia. He thought that if we had methods well adapted to the study of the neuroglia in animals, we should find that the fibres are as distinctly separated from the cells as they are in man. Gliomata may be composed largely of fibres, but may also contain many cells. In a glioma which he had been studying he had

found numerous hypertrophied glia fibres and many cells; some of the later were round and some more or less elongated. He had found certain elements which looked as though they were in a transitional stage from cells to fibres; they were long like fibres and yet seemed to contain a nucleus.

Dr. Spiller spoke of the peculiar sharpness of outline of the gliotic tissue and symmetry of the proliferation in multiple sclerosis, and exhibited specimens illustrating these points. The microscope shows that the proliferated neuroglia is not so sharply separated from the normal tissue as it appears to be to the naked eye. He said he had observed very distinct round-cell infiltration about the vessels within the gliotic patches, which might well raise the question of the relation of disseminated myelitis to disseminated sclerosis.

Dagonet had recently spoken of the hypertrophy of the processes of ependymal cells in general paralysis as a return to embryonic conditions. This would be in accordance with Dr. Flexner's views. There seemed to be no reason why the proliferating glia cells should not assume a primitive form.

Dr. Spiller said he had observed cells in syringomyelia which were evidently very much like those described by Dr. Councilman in epidemic cerebrospinal meningitis. They were quite large round cells, containing two, three or four nuclei and a considerable amount of cytoplasm, and were situated at the outer border of the gliotic tissue. They seemed to be glia cells.

Dr. Flexner thought that the case described by Dr. Sailer might be an instance of a pathological growth of cells of the type of ependymal cells. But if the process were a reparative one rather than a true tumor formation, he would not expect to find either immature or adult ependymal cells in it. His idea was that we should think of neuroglia somewhat in the same way as we are in the habit of regarding the ordinary connective tissues. We distinguish, for example, between fibrosis and fibromatosis, and between granulation tissue and sarcoma. A diffuse growth of fibrous tissue, especially if it is reparative in nature, is not a tumor, not a fibroma. In the same way he would limit the conceptions gliosis and gliomatosis. It remains to be proven that the newly-formed glia, which has nothing to do with tumor, goes through, in the course of its evolution, the earlier stages of its ontogeny, just as growing fibrous tissues often repeat the stages of its embryological development.

The limited time at his command did not permit of a full consideration of the points raised by Dr. Spiller. He was disposed, however, to consider that they agreed in the main questions at issue.

ACROMEGALY AND HYPERTROPHIC PULMONARY
OSTEO-ARTHROPATHY.

Dr. Thayer, of Johns Hopkins University, demonstrated a case of Marie's hypertrophic pulmonary osteo-arthropathy, showing photographs of three similar instances which had been under observation at the Johns Hopkins Hospital. These were accompanied by radiographs illustrating the bones of normal individuals and of two patients with hypertrophic osteo-arthropathy as well as of one instance of acromegaly. The radiographs of the hands and arms showed striking differences between the bones in acromegaly and osteo-arthropathy. In the former there was a general plumpness with an exaggeration of the normal irregularities in outline, and of the points of muscular and tendinous attachment, together with a tendency to roughness and tufting of the bone about the epiphyses. This was especially marked at the ends of the terminal phalanges. In the hands and arms of the patients with osteo-arthropathy a more or less general diaphysial enlargement of the long bones, particularly toward their distal extremities, was to be made out. The terminal phalanges were quite unaffected, and the enlargement of the other bones of the hand was entirely limited to the diaphyses. Especially striking was this enlargement in the metacarpal bones, in some of which the lower thirds of the diaphyses were nearly as large as the distal epiphyses. Autopsies in twenty instances have shown that this enlargement in osteo-arthropathy consists of an ossifying periostitis, limited almost entirely to the diaphyses of the long bones of the hands, feet, arms and legs. Radiographs were then exhibited of an acute case, which showed clearly the newly formed bone as a somewhat irregular shadow along the diaphyses of the long bones of the arm and hand, exactly in the areas of the tenderness and palpable enlargement.

The various theories as to the ultimate cause of this affection were then discussed. It is undeniable that a certain proportion of cases occur without any preceding pulmonary disorder. In a study, however, of 55 typical cases of hypertrophic pulmonary osteo-arthropathy, 43 showed a preceding pulmonary affection. Of the 12 remaining cases, 3 cases followed syphilis, 3 cases followed valvular heart disease, 2 cases followed chronic

diarrhoea, 1 case followed spinal caries, 3 cases followed unknown causes.

The weight of evidence appears to be in favor of a modification of Marie's and Bamberger's theory of the toxic origin of these changes. The majority of the reported cases have followed conditions favorable to the retention of purulent secretions within the economy.

Even if we accept such a theory, however, it is at present impossible to say what the toxic substance or substances may be, how they may arise, or why they should be more frequently present in pulmonary affections than in suppuration elsewhere. The position which we occupy with regard to this affection is not dissimilar to that in which we stand with regard, for instance, to amyloid degeneration. Thus, amyloid degeneration follows commonly chronic suppuration, particularly suppuration of bone, but it is not infrequently found in syphilis where there has been relatively little suppuration, as well as in other cases of chronic cachexia due to malignant disease, or in chronic malarial cachexia, while, lastly, in a certain proportion of cases, no distinct cause can be found. In the same manner we have learned that secondary osteoarthropathies are particularly frequent in chronic suppurative processes connected with the lungs, but the disease has also occurred in several instances of syphilis, 2 instances of chronic diarrhoea and 3 instances of valvular heart disease, and, lastly, occasionally where the condition has seemed to develop spontaneously. In both instances we are led to believe that the process owes its origin to some toxic substance arising within the economy. What this may be we are at present unable to say.

There would appear to be little evidence in favor of the idea that the condition may be due to primary or even secondary changes in the nervous system. There can be no doubt that the name proposed by Massalongo, "secondary hypertrophic osteoarthropathy," is much better than that originally proposed by Marie, but it is a question whether it will be possible to change a term which has come into such general use.

Dr. F. A. Packard said he had been particularly interested in the last case with the evidently recent changes shown by Dr. Thayer, who had been remarkably fortunate in getting the skiagraph at that stage. He was sorry that he had not

brought with him the specimens from a case of pulmonary osteo-arthritis which he reported in 1892. In this case the tibia distinctly shows this thickening of the subperiosteal portion of the bone most marked in, or almost restricted to, its lower half. Dr. Packard had only been able to obtain the tibia in this case, as he was away from home at the time of the necropsy, and the patient's brother had objected to the proposed examination of the rest of the skeleton, notwithstanding the fact that the patient himself had bequeathed his body to Dr. Packard.

Dr. Lewellys F. Barker, of Johns Hopkins University, showed a number of

SPECIMENS ILLUSTRATING THE MEDULLATING CEREBRUM IN HUMAN BEINGS.

The method of preparation was described. The brain of a baby shortly after birth had been hardened in Müller's fluid, imbedded in celloidin, and divided by a laboratory assistant into a series of some four hundred sagittal sections in the Anatomical Laboratory at Baltimore. These were stained by the Weigert-Pal method and mounted in balsam upon ordinary window glass, sheets of mica being used instead of glass cover slips. The glassware and mica needed for such a set of serial sections can be provided at very little expense. The sections are kept in boxes made for holding photographic negatives.

Sections chosen at some twenty levels in the hemisphere were exhibited. Only a few of the fibres are medullated at this stage of development. A portion of the upward continuation of the tegmental radiations, the pyramidal tracts, the optic tracts, and part of the optic radiations already possess myelin sheaths. The exact distribution of the sensory fibres can be easily followed, and the somæsthetic and visual sense areas of the cortex are sharply marked off from other regions. The specimens, as far as they go, are confirmatory of the anatomical discoveries of Flechsig, and illustrate the value of the embryological method for outlining tracts ending and originating in the pallium.

Dr. H. M. Thomas, of Johns Hopkins University, read a paper on

RECURRENT MULTIPLE NEURITIS.

The patient, a merchant, 28 years old, had been at-

tacked on five different occasions by multiple neuritis. These attacks had all commenced in the month of June, and had lasted for from five to six months. The disease had recurred every year, except on one occasion, when the interval had been two years. The symptoms consisted in numbness, swelling and weakness of the feet and legs, with a slight sense of numbness in the hands, except in the last attack, when the hands were also markedly affected. Upon examination, he presented a typical picture of a widespread multiple neuritis.

No etiological factor could be determined. There was no possibility of poisoning by alcohol, nor did there seem to have been any definite exposure to lead or arsenic. The fact that the paralysis had been confined in four attacks to the legs, and that there was no lead line present, was thought to exclude the probability of lead poisoning, although the cistern from which the water at his home was obtained was connected with a lead pipe.

In a review of the literature, very few articles were found in which the recurrence of multiple neuritis had been particularly considered, the papers by Sherwood, Targowla and Sorgo practically completing the list.

Dr. Sherwood's supposition that the first attack of neuritis left the nerves with a lowered power of resistance, and so liable to recurrences, had received a certain amount of confirmation from the result of the autopsy in Sorgo's case. In his case there were marked changes in the blood vessels of the nerves and spinal cord. The patient had died during the third attack of multiple neuritis. In lead poisoning recurrent paralyses are common, when there is a renewed exposure to the lead, and they also do at times occur when there has been no re-exposure (Bernhardt); and it would seem that one attack does predispose to another in the case of this poison.

In alcoholic paralysis, on the contrary, recurrences seem to be rare, and it is remarkable how very generally the cases which are treated are seen in their first attack.

In the records of the Johns Hopkins Hospital there has been but one typical case of alcoholic paralysis which was seen in the second attack. There have, however, been two cases of the ataxic form of alcoholic neuritis which have shown recurrences, and one patient who had had an arsenical neuritis was treated in a second attack brought on by the misuse of alcohol. The recurrence of facial paraly-

sis, although not common, does happen. Among eighty cases there were five second attacks, but in two of these the opposite side was affected, so there were but three definite recurrences.

These cases, although showing that in certain individuals multiple neuritis is likely to recur, do not prove that one attack of the disease predisposes to the second attack. The first attack must be explained as well as the others. The questions of the susceptibility, the resistance and the immunity of the tissues are involved in the answer to this problem. A careful study in this relation of a large number of cases of multiple neuritis would give at least some data upon which to build a plausible hypothesis, but at present no such study has been made, and the question must be left open.

Dr. Wharton Sinkler said that, so far as his experience went, Dr. Thomas' case was unique. From his own observation, he would judge that recurrences in lead and alcoholic neuritis are not common. He recalled only one case in which there was recurrence in lead neuritis, and that was in a house painter of intemperate habits, who had had two attacks. He remembered, also, one case of neuritis in an alcoholic patient, who had had a previous attack. He thought that there is no doubt that alcohol does predispose to attacks of multiple lead neuritis.

Dr. Spiller desired to know whether the recurrent oculomotor paralysis, of which he had seen a good example, might not be thought to have some resemblance to the case just reported.

Dr. Thomas replied that Dr. Mary Sherwood, in her thesis, refers to the cases of recurrent oculomotor paralysis, and gives the literature of the subject. As we do not know the pathology of this disease, the relation which it bears to recurrent multiple neuritis cannot be stated. There are several other forms of recurrent nervous diseases that it would have been interesting to consider had there been time.

Periscope.

With the Assistance of the Following Collaborators:

CHAS. LEWIS ALLEN, M.D., Wash., D.C. R. K. MACALESTER, M.D., N.Y.
J. S. CHRISTISON, M.D., Chicago, Ill. J. K. MITCHELL, M.D., Phila., Pa.
A. FREEMAN, M.D., New York. H. PATRICK, M.D., Chicago, Ill.
S. E. JELLIFFE, M.D., New York. JOSEPH SAILER, M.D., Phila., Pa.
WM. C. KRAUSS, M.D., Buffalo, N.Y. HENRY L. SHIVELY, M.D., N.Y.
W. M. LESZYNSKY, M.D., New York. A. STERNE, M.D., Indianapolis.

ANATOMY.

106. UEBER DEN KERN DES NERVUS ACCESSORIUS (Concerning the Nucleus of the Accessory Nerve). Emil Bunzl-Federn (Monatsschrift für Psychiatrie und Neurologie, 2, 1897, p. 427).

In a number of rabbits the external branch of the accessorius was cut in the neck near its exit from the jugular foramen; in others the roots of the accessorius were cut within the spinal canal. The animals were killed fourteen days later, examination of the larynx having first been made. The movements of the vocal cords were normal. The method of Nissl was employed, as by this stain degenerative changes within a cell are seen soon after its axis cylinder is cut.

Bunzl-Federn finds that the cells of the accessorius nucleus begin in the lateral horn near its base just above the exit of the fifth cervical nerve, and soon assume a position on the lateral and posterior border of the horn; that higher they form two groups more anteriorly located, one of which is more towards the centre; and that still higher the median group disappears and degenerated cells are present in the anterior part of the anterior horn, which later take a more central position within the horn. Degenerated cells were only noticed on the side of the operation. The accessorius nucleus ends just below the lowest part of the hypoglossus nucleus, and extends, therefore, far into the oblongata. Most of the spinal portion and the lowest portion of the oblongatal roots of the accessorius pass into the external branch of this nerve, but a few pass into the vagus. The uppermost fibres of the accessorius nucleus of the oblongata enter the internal branch of the accessorius and thus form part of the vagus. The accessorius, however, has nothing to do with the motor innervation of the larynx, for the vocal cords were not affected after the accessorius roots had been cut, and only in one of many cases was degeneration of accessorius cells found after the recurrent laryngeal nerve had been cut.

SPILLER.

107. UEBER DIE KERNE DER MIT DEN AUGENBEWEGUNGEN IN BEZIEHUNG STEHENDEN NERVEN UND ÜBER DIE VERBINDUNG DERSELBEN UNTER EINANDER (On the Nuclei of the Motor Nerves of the Eyes and their Relations to one another). W. von Bechterew (Arch. f. Anat. u. Entwicklungsgeschichte, 10, 1897, Hefte 5 u. 6).

Bechterew claims four nuclei for the oculo-motorius. The main nucleus is dorsal, situated just beneath the anterior corpora quadrigemina and is paired. There is an unpaired median nucleus, an anterior mesial accessory nucleus and a posterior lateral nucleus. He claims that the upper nucleus, described by Darkschewitsch as the nucleus centralis posticus nervi oculomotorii is in reality a part of the trochlearis.

The different nuclei of the oculo-motorius have numerous association fibres, and he traces fibres of communication between the nuclei of the abducens and those of the oculo-motorius.

JELLIFFE.

108. THE ANATOMICAL CONNECTIONS OF THE FRONTAL LOBES
Joukowski (Revue de Psych., Russian), 1897, Rev. Neur.

The researches of the author were made by the method of extirpation and the study of secondary degenerations. The following conclusions are given.

The frontal lobes are in immediate connection,

1. With the convolutions of the cingulum and the subcallosal fibres by means of fibres of different lengths.

2. With the anterior region of the optic thalamus by means of fibres which pass in the internal capsule.

3. With the substantia nigra, notably its internal portions.

4. With the anterior part of the protuberance, by means of the "fronto-protuberantial" fibres which pass in the internal parts of the cerebral peduncles.

5. Connection between the two frontal lobes is afforded by fibres which pass in the corpus callosum at its anterior end.

6. In the rabbit the connections between the two frontal lobes are reinforced by a fascicle of fibres which passes in the internal capsule and the anterior commissure.

7. There exists a connection between the gyrus fornicatus and the fornix, by means of fibres passing from the former through the corpus callosum to the swelling of the fornix.

VOGEL.

109. ON A MODIFICATION OF THE SUBLIMATE METHOD FOR THE DELINEATION OF NERVOUS TISSUES W. Bevan Lewis (Edinburgh Medical Journal, August, 1897).

The author describes the following:—Pieces of cortex are hardened for from two to three months in Cox's fluid,

5 per cent. solution potass. bichrom.....	20
5 per cent. solution hydrarg. bichlor.....	20
5 per cent. solution potass. chromate.....	16
Water	30-40

They are then washed in alcohol for a half hour, mounted and cut. The sections are brought upon a slide, a drop or two of liq. potassae is added to them, and immediately washed off by inclining the slide, and allowing water from a pipette to flow over the specimen. They are then dehydrated in alcohol, cleared in oil of cloves, and mounted in balsam as usual. The addition of the potash has the effect of bringing out the tissue elements with intense blackness, and reveals the finest details of structure more surely and with greater clearness than the modified sublimate or chrome-silver methods.

ALLEN.

110. ON THE NATURE OF THE WIEGERT-PAL METHOD. J. S. Bolton (Jour. of Anat. and Physiol., 32, 1898, p. 247).

The author presents a very valuable discussion of the whys and wherefores of the Wiegert-Pal methods of staining, with several

serviceable modifications. He concludes his extensive studies thus:—

1. The Weigert-Pal process consists in the incomplete oxidation of a stain from mordanted fibrils containing it as a lake, and bears no necessary relation to the occasional presence of a medullary sheath round these.

2. If an ordinary mordant dye, such as logwood, be used, a metallic salt, to mordant the fibres previously, is necessary.

3. If a basic dye, such as methylene blue, soluble in water, be used, double mordanting with tannic acid, and afterwards with tartar emetic is necessary.

4. The ordinary Weigert-Pal result can be obtained by the use of osmic acid alone, as this forms a jet black lake with hematoxylin in the absence of other metals.

5. Almost as good or equally good results can be obtained by the use of a 2 per cent. solution of iron-alum, ammonium molybdate, ferric chloride, stannous chloride, sodium tungstate, uranium acetate and potash alum; and much inferior results arranged in order of their value can be obtained by the use of similar solutions of nickel sulphate, chrome alum, and the chromates, copper acetate, cobalt nitrate, citrate of bismuth and ammonia, ammonia alum, manganese sulphate and zinc sulphate.

6. Osmic acid and usually iron alum cause a practically equal staining of the medullary sheath and of the axis cylinder, whilst the other metals chiefly cause staining of the axis cylinder, the medullary sheaths being either slightly stained or quite unstained, according to the metal employed.

JELLIFFE.

III. UEBER DIE MARKSCHEIDENBILDUNG DER GEHIRNNERVEN DES MENSCHEN (On the Development of the Myelin Sheath in the Human Brain). A. Westphal (Arch. f. Psych., 29, 1897, p. 474).

The author has studied the development of the myelin sheaths in the brains of newly-born children and in the foetus. He comes to the following general results:—

(a) The Cranial Nerves.*

1. Two groups are here to be distinguished with reference to the development of the medullary sheath. The motor cranial nerves are myelinated at birth, the sensory and mixed nerves are not myelinated, with the exception of the acoustic, which is myelinated early. The optic nerve at its distal end develops latest.

2. The process of myelination proceeds from the central nervous system towards the periphery.

3. With the exception of the optic, the calibre of the nerve fibres increases with age, so that at maturity the calibre may have increased four or five times.

(b) Comparison with the peripheral spinal system.

1. Myelination occurs earlier in the cranial nerves than in the peripheral nervous system.

2. The differences found existing between motor and sensory nerves in the cranial nervous system could not be found for the peripheral spinal system.

JELLIFFE.

PHYSIOLOGY.

112. THE THEORY OF THE MOVEMENT OF THE NEURON AS APPLIED TO NORMAL AND PATHOLOGIC MENTAL AND NERVOUS PROCESSES. F. X. Dercum, M.D. (Gaillard's Med. Jour., 66, 1897, p. 342).

The behavior of the neurons in a case of transient hysterical paralysis may be given as an illustration of the simplest expression

of this theory. What is it that happens in the nervous system when an arm, for instance, is suddenly paralyzed, remains so for a time, and again suddenly recovers its functions? The neurons of the motor area of the cortex present not only protoplasmic extensions directed towards the surface of the cortex, but also an extension downward, which becomes a nerve fibre, but which is, properly speaking, a process of the cell body. This process extends downward through the white matter of the brain and terminates in the cord in a brush-like extremity. By means of this end tuft the cortical cell is brought into relation with the motor nerve cells in the cord. The relation between this end tuft and the nerve cell in the anterior horn of the cord is probably one of contact, and not actual continuity of structure. In a case of hysterical paralysis of the arm, resulting, for instance, from an emotional shock the neurons of the arm centre of the cortex retract their processes in such a way that their end tufts in the spinal cord no longer bear their normal relation to the spinal neurons, and the connection between the cells is broken. The disappearance of the paralysis is due to the extension of the processes previously retracted—that is, the end tufts resume their normal relations with the spinal neurons and function is re-established. Sleep has been commonly attributed to an anæmia of the brain, notwithstanding the fact that true cerebral anæmia, such as follows great loss of blood, is attended with insomnia. The movements of the neurons will account for sleep. When functionally active they must be in relation with one another and their processes either in contact or nearly so. Evidently this condition is a requisite of consciousness. When the nerve cells are exhausted by fatigue, their volume and contents diminish, as we have every reason to infer from the experiments of Hodge. In sleep the neurons have their processes retracted; in consciousness their processes are extended. This theory also enables us to explain pathologic unconsciousness, such, for instance, as attends cerebral concussion. A man receives a blow upon the head; the neurons suddenly retract their processes and unconsciousness results. It is extremely probable that the unconsciousness of chloroform or ether anæsthesia, or the sleep produced by drugs may be explained similarly. A sequence of sound vibrations impinging upon the peripheral auditory neurons produces a change that affects the relations which their centripetal processes bear to the nerve cells of the auditory nuclei of the medulla, and secondarily to the neurons in the auditory area of the cortex. It is probable that the effect produced upon the latter is such as to cause them to retract, extend or otherwise move their processes. The same sequence of sound vibrations must always produce the same changes in the cortical neurons. The physiology of conception evidently consists in the reforming among the neurons of an old combination—that is, the combination, the same as, or similar to, what was originally produced by a physical impression upon the organs of sense, but which is now produced by some other agency. To explain the phenomena of memory, we will say that a sequence of sound vibrations has impinged upon the peripheral auditory neurons and in turn has caused the neurons of the auditory cortex to move their processes. Other things equal, the degree of the excitation depends upon the intensity of the physical impact upon the auditory apparatus. Evidently the number of auditory neurons aroused into action varies greatly, and the movement does not cease in the auditory area of the cortex, but is diffused among distant neighboring areas. Suppose a sequence of sound vibrations has caused the auditory neurons to assume new relations with one another. These relations will depend largely, whether or not a similar sequence of impressions has passed

through those neurons before. If so, the old combinations will be reformed, and as a corollary, there follows the recognition by the ego of the sounds as something heard before. Sequence of thought may also be explained by the movements of the neurons. If they are constantly changing from the hour of awakening and responding to stimuli from without by changes in their relation to one another, it follows that the sequence of thought depends upon the sequence of these changes. Herein also lies the explanation of the continuity of thought. This theory is also applicable to various pathologic conditions. An hallucination is the spontaneous formation of an old combination among the neurons representing a former perception or conception as the result of pathologic conditions and not in response to normal stimuli from within, such as preceding and connected trains of thought, or other normal psychic processes. The combination representing an hallucination reacts in such a manner upon the general neurons of the cortex as to give rise to the belief in the reality of the manifestation. An illusion is such a faulty combination of the neurons as to lead to an imperfect recognition by the ego of the object perceived.

It is probable that the explanation of the pathology of a delusion is to be sought in disease of the structure of the neuron and especially of its processes. All the symptoms presented by hysteria, and even the convulsions, can be explained by this theory. The mobility of the neuron is the only theory that affords a rational explanation of hypnotism. Hypnotism is a partial sleep, in which only a portion of the neurons have their processes retracted. When hypnosis is induced by staring fixedly at an object, while suggestion of sleep is made, it happens by reason of the effort of visual and auditory attention that the neurons of the corresponding regions in the cortex are thrown into certain relations with one another, corresponding first to the action upon the ocular apparatus and, second, to the words of the operator. Soon, by reason of visual fatigue and under suggestion of sleep, the neurons of the visual area retract their processes, and the partial sleep of hypnosis begins. Gradually it grows deeper, by reason of the same influence, the other neurons of the cortex gradually retract their processes, with the single exception of those by means of which the verbal suggestion of sleep has been received, namely the auditory channels.

FREEMAN.

113. SULLA FISIOLOGIA DEI TALAMI OTTICI (The Physiology of the Optic Thalamus). Lo Monoco (Riv. di patol. nerv. e ment., 2, 1897, No. 8).

The author reports the results of three partial excisions of the optic thalamus in dogs, the operation having been performed in the manner detailed by him in a former communication. In two cases only the inner portion; in the third, both outer and inner portions of the dorsal part of the thalamus of one side were removed. Blindness, diminution of sensibility and impairment of muscular power set in on the side opposite to that of the injury, but disappeared after a period of about four weeks.

The conclusion of the author is that the optic thalamus is the seat of a visual perception centre similar to the one found in the cortex, but with the difference that its removal does not cause either dilatation or loss of contractile power of the pupil. The relation of the thalamus to sensation and mobility is similar to that of the cortical centres.

JELLIFFE.

114. THE FLUTTERING PRODUCED BY THE JUXTAPOSITION OF CERTAIN COLORS AND OF BLACK AND WHITE. W. A. Holden. (*Archives of Ophthalmology*, 27, 1898, p. 1.)

In a series of ingeniously devised experiments the author shows that "when two colors of nearly equal luminosity are juxtaposed, one color will seem to dart over the other color, and the margin between them will appear constantly to shift as the eyes or colors are moved. This fluttering is due to the negative after-images of each color being projected upon the other color, and it is seen best when the two colors are nearly equal in luminosity, because an after-image of an object arises most readily when surrounding objects are of the same luminosity, and also because after-images are perceived most readily when projected on a ground of the same luminosity. An after-image of short duration gives rise to an appearance of flashing. An after-image of longer duration projected upon a background of nearly the same color intensifies that color, and gives rise to an appearance of glowing. The after-images, appearing and fading away, and shifting with each movement, give rise to the appearance of fluttering.

Different colors on a dark ground appear to stand out in different degrees of relief, and this has been confounded by some authors with the phenomenon of fluttering. But apparent relief is entirely independent of hue, and depends solely upon relations of luminosity; it being greater the greater the difference in luminosity between object and background, while fluttering is most apparent when the difference in luminosity is least.

Black on a purely white ground readily gives rise to white after-images, which cause either flashing or glowing, according to their duration, and black objects on white thus appear to flutter as do juxtaposed colors of equal luminosity. Such after-images arising from the cumulative fatigue of the retina in reading successive lines of print give rise to much of the discomfort experienced in reading badly printed pages, and the printer's aim should be to compose a page in which the disturbing effects of these after-images are reduced to a minimum."

JELLIFFE.

PATHOLOGY.

115. WEITERE BEITRÄGE ZUR PATHOLOGIE DER NERVENZELLE. III. 1. UEBER GANGLIENZELL-VERÄNDERUNGEN BEI KÜNSTLICHER STEIGERUNG DER EIGENWÄRME. H. Moxter. 2. UEBER VERÄNDERUNGEN DER NERVENZELLEN IM FIEBER. Goldscheider and E. Flatau. 3. UEBER DIE VERÄNDERUNG DER MENSCHLICHEN NERVENZELLE BEIM FIEBER. S. Goldscheider and F. Brasch. (*Fortschritte der Medicin*, 16, 1898, p. 121).

A series of observations are made in these short contributions. The first series was made upon rabbits; these were trephined, and, after recovery, were exposed to temperatures ranging from 39° to 41° C., with the following results:

(1.) Changes in the anterior horn cells were found only after 22½ hours' exposure to a temperature of between 40.5° and 41.5° C.

(2.) By exposure to an intermittent temperature of between 38° and 41° C. for several days no cell changes were induced.

(3.) After an exposure of 23 hours to temperatures between 39.2° and 40.7° C. no changes were apparent.

These observations confirm those previously made by Goldscheider and Flatau in a previous number of the *Fortschritte der Medicin*.

The second contribution takes up the question from the human standpoint. The authors try to show the effect of fever upon the ganglion cells. In six cases, in which the fever varied from 37° to 39.9° C., the cords were studied by means of the Nissl methods. They found that numbers of the cells of the anterior horns were enlarged, and that they stained less readily. There was marked chromatolysis throughout. The granules of the protoplasmic processes were absent also. The nucleus remained in the centre of the cell and showed no marked deviations from the normal. All levels of the cord were affected, the cells of the anterior and posterior horns as well as those of Clarke's columns.

JELLIFFE.

116. LESIONS IN HEREDITARY CHOREA. Dr. Lannois (Medical Week, August 13th, 1897).

At the French Congress of Alienists and Neurologists the author reported post-mortem examinations of two female patients suffering with hereditary chorea. In both there was pachymeningitis, recent hæmatoma, and very marked atrophy of the brain. Under the microscope the lesion appeared to consist very distinctly of an infiltration of small round cells made up almost exclusively of a large nucleus, found in small numbers in the area of polygonal cells, increased in the area of small pyramidal cells, and reaching its full development in the area of large pyramidal cells. These round cells were also met with in the subjacent white substance. They were mostly arranged in groups around the pyramidal cell, invading its lymphatic space, or around the vessels, and inside or around the peri-vascular sheath. The spinal cord appeared also to be slightly diseased in the descending columns, in the centro-lateral regions, and in the direct cerebellar tract. The author considers that the motor as well as the mental disturbances of such cases are fully accounted for by irritation of the neurons from the invasion of the nuclei into the pericellular sheaths.

MITCHELL.

117. BEITRÄGE ZUR LEHRE VOM WESEN DER HUNTINGTONSCHEN CHOREA (Contributions to the Study of Huntington's Chorea). F. C. Facklam (Archiv. f. Psychiatrie, 30, 1898, p. 137).

The present dissertation is a careful and extensive contribution to our knowledge of Huntington's Chorea, both from the clinical and pathological standpoints.

The author gives a historical sketch of the two types of chorea, so-called, and the histories, in extenso, of eight cases of Huntington's syndrome.

From the pathological point of view the author is of the opinion that the disease is exclusively confined to the cortex, and that the changes are of the chronic diffuse encephalitis type though differing from the "disseminated" type of Oppenheim, or the "diffuse encephalitis" described by Kalischer. The author sums up with a brief discussion of the differential diagnosis, in which little new appears. He states—

1. That whereas Huntington's chorea is a disease of adults, Sydenham's chorea is found mainly in childhood.

2. Huntington's chorea almost invariably shows an hereditary history, whereas according to the greater number of the more recent investigators, chorea minor is to be looked at in the light of an infectious disease.

3. Huntington's chorea is invariably a chronic affection and non-curable, whereas chorea minor is acute and readily curable, the sub-acute or chronic cases usually recovering.

4. The main point of distinction is to be seen in the mental symptoms. Huntington's chorea being markedly chronic leads to mental deterioration and ultimately to dementia, whereas the psychical changes in chorea minor are rarely marked.

The feature in common, that of the choreic movements, is one in name more than in fact, for the movements are essentially different. The motions in Huntington's chorea being, as a rule, slow and less active than in chorea minor, although both have a like place of origin, the cerebral cortex.

Huntington's chorea is to be regarded as an individual disease, distinct and independent of Sydenham's chorea.

Most of which statements are found in American text books.

The article is concluded by a fairly complete bibliography and is illustrated by one lithographic plate, showing the changes in the blood vessels and lymphatics.

JELLIFFE.

CLINICAL NEUROLOGY.

118. DE LA CHORÉE VARIABLE OU POLYMORPHE (Variable or Polymorphous Chorea). G. Patry (Gaz. hebdomadaire de médecine et chirurgie, 2, 1897, p. 105).

Expanding the observations and following the plan of Brissaud, the author in his thesis (Paris, 1897), endeavors to differentiate a form of chorea to which he gives the above name. The special definition is as follows: "It is a chorea which has neither uniformity in its actual symptoms, regularity in its evolution, nor constancy in its duration. It goes and comes alternately, increases and diminishes, ceases suddenly, and begins again with equal suddenness, to disappear once more. The movements are sometimes sharp, and sometimes slow, and without any preponderating localization. It is more common in the degenerate type of individual, that is, in those presenting the physical stigmata of degeneration, and whose psychical equilibrium may, therefore, be said to be unstable. It occurs at an age somewhat greater than that at which ordinary chorea appears, being more commonly observed after puberty."

After a discussion of etiology and pathogeny, he goes on with his observations on the symptomatology; as it occurs in degenerates, the usual psychical lack of development or inequality of development is found. The ordinary signs of unstable nervous and mental condition were present in many of his cases—hysteria, epilepsy, hallucinations. All the patients complain of vague pains of varying situation and degree, not unlike those common in neurasthenic cases. The movements, sometimes large, sometimes small, vary in amplitude and frequency of occurrence from one minute to the next, and from one day to the next.

They changed their locality as well as their intensity, become general or limited, or even disappear without a hint of any reason for the changes. The final capital symptom is the very decided inhibitory influence which patients can exercise over the movements.

As may be supposed from these statements, the disease is of indeterminate duration, and the tendency to disappear, and the tendency to relapse make prognosis very uncertain. The treatment he considers purely moral, that is to say, he endeavors to point out to patients the possibility of control, and the undesirability of making a spectacle of themselves, and thus encourages them to use the undoubted power of inhibiting the movements which he has observed.

MITCHELL.

119. A CLINICAL RE-EXAMINATION OF THE MOTOR SYMPTOMS OF CHOREA. S. W. Mitchell and J. H. W. Rhein (Phil. Med. Jour., 1, 1898, p. 153).

The histories of some thirty cases are presented in this short study of the types of movements to be distinguished in chorea. They are grouped so as to present four kinds of movement. These types are—

1. Cases of chorea which show, some at one stage of the disease, some throughout their course, an absence of movement during rest, requiring muscular action to develop what may be either mild or severe choreiform movements.

2. There are cases in which the movements are continuous during rest, but become greatly increased on intentional effort.

3. There are cases with severe choreiform movements which disappear entirely when muscular acts are performed.

4. In some cases the movements seem to be unaltered by voluntary muscular efforts.

5. There are cases which present during their course, at different times, more than one of the types described.

JELLIFFE.

120. CHOREIC EMBOLISM S. S. Adams (Annals of Gynæcology and Pædiatry, 10, 1897, p. 239).

The rarity of the case cited induced the author to report it.

Arthur D., aged 7 years and eleven months, white, was admitted to the Children's Hospital (Washington, D. C.), June, 1896. Excepting tuberculosis in his maternal grandfather, his family history was good. The boy had been very studious, working well into the night. He was well prior to February, 1896, when he was taken ill with measles. He apparently recovered from the attack, and while seemingly in good health, he was suddenly seized with vertigo, photophobia, and inability to walk. He complained of no pain. His eyes became crossed, and his muscular inability grew manifestly worse. His present condition is fair. He sleeps well, though very sensitive and peevish. There is pronounced strabismus in the right eye, otherwise the eyes are normal. The left angle of the mouth droops, and when the tongue is protruded, it deviates to the left. Articulation is slow, indistinct, nasal, but questions are answered intelligently. There is ptosis and paralysis of the left facial muscles. The right arm is normal, but in the left there is neither motion nor sensation of ordinary touch, but a pin thrust is felt, and if a strong irritant be applied to the fingers, he locates it accurately. Reflex motion is only slightly impaired. The left lower extremity is only partially paretic, but seems to be increasing. The patellar reflex is slight on the affected side, and there is no clonus, but there is constant formication. There is no profuse sweat at night. Other systems normal. On further and later examination a slight choreic twitch on the right side was discovered. It was also found that he had an attack of chorea sometime before but had recovered. The reflexes were absent on the left side, and the surface temperature was lower than on the right. He could not walk without assistance. While in some respects the case resembled one of posthemiplegic chorea, yet the previous attack of chorea made the differential diagnosis easy and justifiable. The speedy and complete restoration to health added further corroborative evidence to the correctness of the diagnosis.

ABRAHAMS.

121. SEBORRHOEA NIGRICANS (An Unusual Hysterical Disorder). J. K. Mitchell (Phila. Med. Jour., 1, 1898, p. 117).

A peculiar case of intense pigmentation of the region about the eyes is here reported and illustrated. The line of pigmentation ex-

tended from $1\frac{1}{8}$ to $1\frac{3}{8}$ inches below the nasal margin of the lid, the color always being less on the upper lids than on the lower.

The pigment, which imparted a sooty-like sense to the finger, could be removed in part by appropriate methods, but such removal was but of temporary service as the color returned in a few hours. After some time the pigmentation ceased, but some months later again returned at a time when the patient was greatly fatigued and run down. The case is considered one of hysteria, and an historical resumé of the subject is included in this report. A microscopical and bacteriological examination of the pigment removed was practically negative.

JELLIFFE.

122. SUR UN CAS D'HEMIPLÉGIE HYSTÉRIQUE ACCOMPAGNÉE D'ATROPHIE (Hysterical Paralysis with Atrophy). Lyonnet and Bonne (Lyon Médical, 86, 1897, p. 286).

As nearly as one may judge from the report, this is quite a typical case of hysterical hemiplegia, occurring in a nervous young woman of 20 years. The trouble had existed for 8 months, had come on gradually, and at one time the patient could not "budge" the left arm or leg. The character of the patient, the mode of onset, the gait, the distribution of the paralysis (face exempt), the intensity and distribution of the anesthesia, as well as absence of all indications of organic disease, indicated hysteria as the cause of the paralysis. In the paralyzed members there was a generalized and rather uniform wasting of the muscles, without electric change. The upper arms showed a difference in circumference of $1\frac{1}{2}$ centimeters, the forearms a difference of $2\frac{1}{2}$ centimeters, and the thighs and legs 3 and $1\frac{1}{2}$ centimeters respectively.

PATRICK.

123. CONTRACTURE HYSTÉRIQUE (Hysterical Contracture). Dejerine (La Med. Moderne, 8, 1898, p. 38).

The author exhibited at the Salpêtrière a case which brought up the general question of hysterical contracture. The accompanying stigmata generally suffice to differentiate the neurosis, but the origin is often sufficiently difficult to establish. In the case described Déjerine found a contracture of the adductor muscles of the thighs in a young girl, consequent upon an attempted assault, of such intensity that not only was walking impossible, but the utmost effort could not separate the limbs in the smallest degree. The contracture yielded entirely under chloroform. A case of the opposite condition was also shown in which the neuropathic character of the disorder was less evident. A girl of eighteen walked with the limbs separated to their utmost extent, the thighs being in the most extreme abduction. The articulations showed nothing abnormal; the freest movements, except in adduction, could be obtained; there was no pain nor stiffness. The hypothesis of ankylosis of the coxo-femoral articulations was not, therefore, acceptable. There was decided contraction of the abductor muscles, and the question of its origin arose. The patient stated that six months before she had had pains in the joints, and her report was that her physician had said that she had albuminuria. It was, therefore, possible that an infection had some relation to the beginning of the trouble.

Déjerine, however, thought this had been the provocation of the condition, not the cause. The patient was really an hysteric, and her difficulty due to this neurosis. He, therefore, ordered treatment "by isolation and suggestion," with what result is not reported.

MITCHELL.

124. PARALYSIES TRANSITOIRES D'ORIGINE CARDIAQUE (Transitory Paralysis of Cardiac Origin). MM. Achard and Léopold Levi (La Méd. Moderne, 7, 1897, p. 656).

MM. Achard and Léopold Levi reported their observations of two cases where, apart from paralysis due to gross cerebral lesions, transitory palsies occurred. The first case was a woman, who in the course of an attack of asystole had an inferior facial paralysis which lasted four days, and then completely disappeared. She was seized some days later with hemiplegia of the opposite side, and died. Histological examination showed only an active congestion and slight oedema, specially marked at the base of the first frontal convolution. The second case was that of a patient with mitral stenosis who had paralysis of the limbs of one side, and a facial palsy of the peripheral type, together with paralysis of the sixth nerve, upon the other side. This paralysis lasted for four days. These cases are related to those seen in the course of uraemia and hepatic intoxication, in which the authors say that they have also observed isolated and transitory facial palsies.

MITCHELL.

125. A CASE OF PARALYTIC CHOREA. M. V. Ball (Phila. Med. Jour., 1, 1898, p. 299).

A case is here reported of a child of seven, who suddenly developed pain in her various joints, followed by rapid and irregular respiration and heart's action. At the same time fine choreiform movements developed about the lips and arms. There was slight bronchitic involvement which soon disappeared, but the chorea persisted and increased. All the limbs were involved, and talking was interfered with. At the end of the week paralysis of the limbs developed, and the child lay passive, unable to do anything. Arsenic was employed, and at the end of two weeks there was an improvement, and at the end of six weeks recovery was complete, save for the persistence of a heart lesion.

JELLIFFE.

126. A PECULIAR FORM OF TIC CONVULSIF. F. G. Finlay (The Montreal Medical Journal, 25, 1897, No. 9).

The author reports two brothers affected with a peculiar form of family tic convulsif with nocturnal exacerbations and epileptic attacks. The patient's mother suffered from chorea in childhood, and insanity of pregnancy. The family is decidedly neuropathic. Previous to the onset of the convulsive movements Jean Degan, 23 years old, was impelled to execute any sudden command, even to render a person insensible. His trouble dated six years back. At present the patient is dull and stupid. He is muscularly well developed. Every few seconds a single twitching movement of one or the other side of the mouth, or a single similar contraction of the fingers of one or the other hand is observed. The movements are slight in degree, and apparently unaffected by his attention being drawn to them.

In addition to these twitching movements, marked jerking, inco-ordinate clonic movements are induced when he attempts to perform any action. During sleep both the twitching and the jerking inco-ordinate movements continue and are, indeed, much increased. The motor power is slightly diminished; sensation is unaffected; the knee-jerks are increased, and the pupils react well to light and accommodation. The optic disk is normal, and there is no nystagmus. One night he had several convulsive attacks which lasted about half an hour and were accompanied by sounds like the yelping of a dog.

The second case, Alex Degan, 20 years, closely resembles the first. In both cases the epileptic attacks followed the onset of the convulsive movements, and in view of the neurotic family history may be looked upon as expressions of hereditary nervous degeneration.

ABRAHAM.

PSYCHOLOGY AND PSYCHIATRY.

127. A CONTRIBUTION TO THE STUDY OF ILLUSIONS. Frederick E. Bolton (*American Journal of Psychology*, 9, 1898, p. 67).

The author experimented on twenty-five persons and found they underestimated the size of circles and overestimated the size of squares as a rule.

CHRISTISON.

128. A STUDY OF IMAGINATIONS. George V. Dearborn (*American Journal of Psychology*, 9, 1898, p. 181).

Dr. Dearborn tested a company of sixteen persons, comprising Harvard professors and their wives and some students from the Psychological Laboratory. Chance figures formed by ink drops, which had been pressed between slips of paper, were one by one presented for the imagination to decide what they resembled or suggested. Several of the tests showed that no two persons thought alike on the resemblance of the figures, and in only one test, which was, a decidedly human-like figure, did as much as 40 per cent. of the company correspond. The answers were made in writing.

CHRISTISON.

129. SOME EFFECTS OF SIZE ON JUDGMENTS OF WEIGHT. H. R. Wolfe (*Psychological Rev.*, 5, 1898, p. 23).

The author experimented extensively on students, both male and female, with pieces of lead and blocks of light wood, the ratio of weight to size being 1 to 25. His experiments were repeated during several years, and showed that the wood blocks were invariably judged to be lighter than they really were, and the lead pieces heavier than they actually were. The illusion was greater for women than for men, and greater for small than for large weights.

The variation for individuals was found to be immense, but, by eliminating 10 per cent. of the subjects—those known to be abnormal—the variation is greatly reduced. With both men and women the ratio of wood to lead at first increases with the weight, then decreases more rapidly, till for the heaviest weight the ratio is about two-thirds as great as for the lightest weights. For example: "The men find 4.7 grams of lead equal to 15.5 grams of wood. The women find 3.1 grams of lead equal to 15.5 grams of wood. The men think 229.2 grams of lead feel as heavy as 525 grams of wood, while the women select lead weighing only 145.2 grams as equivalent to 525 grams of wood."

"If we ascribe one-half of the error to each substance, we find that, in comparing lead and wood weights, men estimate the lead at about twice its actual weight, and that they estimate the wood at about two-thirds its actual weight. Under the same conditions, women estimate lead at more than three times its real value and wood at less than three-fifths its real value."

Methods of lifting did not seem to exert any appreciable influence. As a rule, the lead was lifted in the palm of the hand and the wood between the thumb and fingers. "If the weights be suspended by a cord held between the fingers, so as to give no intimation as to which is lead or wood, and the eyes be closed and all other means of determining size be excluded, the judgment immediately improves, until the error amounts to less than one-tenth of the weight; while, with a knowledge of the relative size, the error may rise to three or even ten times the weight."

Experiments with paper bags filled with cotton or air, and also with brass cylinders, contain the same factors and show similar results. The size of the error of an individual is a function of his personality.

CHRISTISON.

THERAPY.

130. THE VALUE OF ARSENIC AND BELLADONNA IN THE TREATMENT OF CHOREA. W. Overend (Lancet, 2, 1897, p. 248).

1. Belladonna appears to be most beneficial in recent cases, and its influence is sometimes very marked in severe forms.

2. In obviously rheumatic cases arsenic in large doses may be given a trial, or may be combined with belladonna from the first. Belladonna may act by diminishing the excitability of the nerve centres or by imparting an improved tone to their vascular supply.

3. In the wards of a hospital it is perfectly justifiable to give to a child as much as thirty minims or more of the tincture of belladonna every four hours for ten days, or even longer. Certain precautions are necessary. The patient should be kept in bed, and the urine should be daily measured. Small doses of potassium acetate may be added if it becomes much diminished, or if the eyelids show any puffiness. In one child nocturnal incontinence occurred, and the dose was lessened. The occurrence of the papular erythema, which leaves raised circular lumps for a while, does not necessitate any diminution of the dose. Dryness of the throat and swelling of the parotids, should they occur, are merely temporary. The influence of the belladonna makes itself felt after about four days. Should no visible improvement occur before the tenth day, it would be useless to continue with it. But in eight severe cases treated belladonna was of benefit, and is certainly worthy of further trial. As soon as the movements become trivial, or occur only during exertion, it is better to omit the belladonna, to commence massage of the affected muscles, and administer cod-liver oil and syrup of phosphate of iron or other tonics. The arsenic may be continued for a week or longer.

JELLIFFE.

131. TRAITEMENT CHIRURGICAL DE LA NÉURALGIE FACIALE (Surgical Treatment of Facial Neuralgia). P. Mouclair (La Presse Médicale, 5, 1897, p. 261).

The author gives an excellent résumé of this subject. Considering first the causes of facial neuralgia, he then proceeds to give in outline the various operative procedures for its relief. As illustrating the results of extra cranial nerve section, the analysis, made by Bessard in 1882 of 244 cases, with 7 cures, 23 ameliorations, and 7 deaths, is not of much value, as the operations were done before the days of anti-septic surgery. Better are, 10 cases, with 6 cures, 2 ameliorations, and 2 failures, reported by Le Deutu in 1894.

To show the results of the intracranial operation of removing the Gasserian ganglion, the author combines 3 cases collected by Beck in 1895 with 19 others from various sources.

There were 30 cases of the Hartley-Krause operation with 4 deaths, and 20 cases of the operation of W. Rose with 5 deaths. Removal of the Gasserian ganglion seems to give an almost certain cure. It does not cause serious trophic disturbance in the eye or on the face, and mastication can still be accomplished. There is diminution of tactile sense and of smell and taste, on the operated side. While it is a formidable operation, and should only be undertaken by a skilled surgeon, the suffering in facial neuralgia is so intense as to justify its performance in cases not relieved by change or by intra-cranial nerve section.

The Hartley-Krause operation is regarded as the preferable one.

C. L. ALLEN.

Book Reviews.

LES AFFECTIONS NERVEUSES SYSTÉMATIQUES ET LA THÉORIE DES NEURONES. By J. M. Gerest. J. B. Baillière et Fils, Paris, 1898.

The writer attempts to explain the various affections of the nervous system on the neuron theory. He divides his book into two parts; in the first he treats briefly the diseases of the motor neurons, and in the second, those of the sensory. The neuron theory probably affords the simplest means of explaining nervous diseases, but there is some danger in making use of this. Man loves sharply defined pictures, and there is a tendency ever present to separate diseases in a way that nature does not always permit. Especially is this true in regard to the maladies of the nervous system. We constantly overlook the transitional forms, and forget that where a diagnosis depends chiefly on the location of the lesion, as is the case in many nervous diseases, we must expect to find borderline symptoms. When we employ the neuron theory, we are very liable to consider the affections of the central neuron as distinct from those of the peripheral; or those of the sensory from those of the motor. We are beginning to believe, however, that any change in the central tract or in the sensory peripheral fibre, affects the condition more or less of the peripheral motor neuron.

Dr. Gerest has treated the subject in a skilful manner, and his book will probably make the affections of the nervous system more comprehensible to many.

SPILLER.

DIE BEDEUTUNG DER AUGENSTÖRUNGEN FÜR DIE DIAGNOSE DER HIRN- UND RÜCKENMARKS-KRANKHEITEN. FÜR AERZTE, BESONDERS NEUROLOGEN UND OPHTHALMOLOGEN. Von Dr. Otto Schwarz, Privatdocent an der Universität, Leipzig. S. Karger, Berlin, 1898.

The author here presents in a short and practical manner those disturbances in the functions of the eye which are of importance in the diagnosis of diseases of the brain and spinal cord. He lays particular stress upon the methods of examination, and then discusses the diseases of the brain and cord singly, laying particular weight upon the ophthalmoscopic examination. The book occupies the middle ground between the two special fields, and will undoubtedly prove of service to the neurologist and the general practitioner.

JELLIFFE.

LES LOCALIZATIONS DES FONCTIONS MOTRICES DE LA MOELLE ÉPINIÈRE. Par le Dr. Fritz Sano. Brussels, 1898.

The present essay is a reprint from the "Annales de la Société Médico-Chirurgicale d'Anvers." It contains much of interest to the neurologist, though the facts have been fairly well given in most of our best modern text books. Some new observations are recorded, and the paper represent a careful summary of our present knowledge of spinal cord localisations.

JELLIFFE.

L'HYSTÉRIE AUX XVII. ET XVIII. SIÈCLES. ÉTUDE HISTORIQUE ET BIBLIOGRAPHIQUE. Par Mme. G. Abricossouff, Docteur en médecine de la Faculté de Paris, G. Steinheil. Paris, 1897.

To those interested in the historical aspects of mental epidemics this well-prepared brochure will prove of great service. The author has given an exceedingly good review of the literature and has done a large amount of literary research which will be of benefit to those following in similar lines of work. He has shown that the disease was well recognized in these centuries and has brought together many obscure writings which, to consult in the original, would involve an immense amount of labor.

JELLIFFE.

THE PSYCHOLOGY OF THE EMOTIONS. By Th. Ribot. London, Walter Scott; New York, Charles Scribner's Sons. 1897.

This important work, by one of the foremost of modern psychologists, can best be delineated in the short space at our command by stating the author's main position with reference to the affective life. Ribot is one of the advanced school of physiological or biological psychologists. There is little of the metaphysician or schoolman about him. He is thoroughly scientific, and regards his subject as much as possible as among objective phenomena to be analysed and explained. This gives his work great value to the neurologist as well as to the psychologist. All such readers must feel instinctively from the first pages that Ribot has contributed to the real science both of neurology and of psychology.

For Ribot the origin of the emotional life coincides with the very origin of the physiological life. Every emotion is represented by—or reducible to—first, a motor element, and second, a mixed element of pain, pleasure or indifference. This motor element is, however, the primordial constituent of the affective life. It represents the *impulses* that are inherent in organic matter; and Ribot finds its origin in even the lowest protoplasmic cells, in which such impulses are but the workings of a chemico-biological life. In this primitive stage these impulses of attraction and repulsion represent merely the blind unconscious or sub-conscious activities of nutrition, and are not far removed above the mere chemical affinities and incompatibilities of inorganic substances. It is only later in the evolution of the animal organism that there is superadded to these impulses the conscious element of pleasure and pain, and still later perhaps the conscious element of intellection.

Starting with this profound conception of the affective life, Ribot, by a masterly analysis, reduces the primitive emotions to five in number. These are fear, anger, affection, the self-feeling and the sexual emotion. These are named in the order of their appearance—and the dates of their appearance in the individual, as can readily be seen, are by no means coincident.

From this view of the affective life it naturally follows that this life is the first in the order of development of the psychic functions. It is deeper seated, and in every sense more primitive and essential in the nerve-cell than the mere intellectual life. Thus its importance appears vastly greater from the biological standpoint than it has ever appeared to the dogmatic or metaphysical school of psychologists. From the logical deductions of this position Ribot does not shrink for a moment.

His logical position in his own words is this: There is a pure and autonomous life of feeling, independent of the intellectual life and having its cause below, in the variations of the coenaesthesia, which is itself the resultant and concert of vital actions. Internal sensations, i. e. such as arise from the processes and needs of nutrition, rather than external sensations, are the sources of this emotional life. From this it follows for Ribot that there is an emotional state unconnected with an intellectual state. Hence Ribot places himself in direct opposition to

the Intellectualists, who contend that there can be no pure emotional state without an intellectual state. For these latter emotion has always been secondary to intellection; it has merely existed, to quote Ribot, as a sort of efflorescence on the intellectual life. We have seen, from the brief statement already made, how completely the biological method of Ribot reverses this position, and, seemingly with great accuracy of observation and analysis, assigns to the affective or emotional life an existence not only independent of, but prior to, the intellectual life.

We have nowhere seen this purely biological position so strongly and, we must say, so convincingly stated as by Ribot. Its immense importance to the scientist, and especially to the pathologist and alienist, can readily be seen. Ribot himself is thoroughly alive to its bearings on psychology and neurology, and draws largely from these specialties for argument and illustration. If the emotional life is so truly fundamental and primitive in the evolution of the brain-cell, then we may be prepared to accept the most advanced teachings of those who see in some forms of insanity and the neuroses the most radical changes in the affective life quite independent of disorder of the intelligence.

For ourself we must say that, while feeling the force of Ribot's subtle analysis and plausible argument, we cannot quite divest our mind of the suspicion that after all the argument may be too analytical: that we may be under the spell of a master dialectician. When we attempt in either an objective or subjective study to dissociate the intellect and the emotions, we are always driven back to at least a tentative position from which the thing seems impracticable and even inconceivable. Pure emotion, without ideation or representation, at least in the adult mind, is an abstraction. We say this with great deference to the author whom we are reviewing—for his biological argument must appeal strongly to every scientist. We merely ask, is it not possible that Ribot has too much ignored the evolution of the intellectual life? Even in the primordial and instinctive impulses of the very lowest forms of life is there not in every such act some element also of representation, so feeble perhaps as to defy analysis, but forming an integral part of every true psychic act?

In his discussion of pleasure and pain Ribot's biological method is conspicuous. He bases this study on a careful inspection of the anatomy and physiology of pain, and its tracts of transmission in the central nervous system. His own position with reference to some of the complex problems of pain and pleasure is clear, although he frankly confesses that he leaves the subject itself in some points obscure. This is unavoidable as yet by any psychologist. For him pleasure and pain are not emotions: the fact that they were ever regarded as such being due to the great perversion and misappropriation of terms, so common in psychological writing. Pain is merely an epiphenomenon, and not an essential part of an emotion. Ribot proves this from actual observation. Hence his position seems to be that pain is a mere quality of a mental state. The same emotion may be painful or pleasurable at different times. This independence of the pain-phenomenon is rejected by the Intellectualists: and here, again, Ribot takes a stand at variance with tradition—a stand to which his method leads him. From this stand, too, he rejects the doctrine that pain is in itself a sensation—although he gives due weight to the evidence from pathology, as in syringomyelia, etc., that pain and common sensation may be dissociated, and, hence, appear to have different tracts in the spinal cord. His teaching, as already said, seems to lead to the quale theory—which he discusses but does not unreservedly accept. He rather views with favor the theory of Oppenheimer that pain is caused by chemical changes in the tissues, i. e., the action of autogenous toxins. But we do not see how this throws light upon the real psychology of the subject. It merely

removes the etiology one step inwards. Finally, Ribot makes clear the point that pain is essentially the same thing, whether physical or moral: these forms differ from each other merely in the fact that the former is connected with a sensation, the latter with some form of representation, i. e., an image or idea. The conditions of pain and pleasure seem to be the lowering and heightening respectively of the vital energy. The question is thus reduced to a physiological one.

Ribot's work is divided into two parts. The first is devoted to general psychology—the basis for which we have attempted to state in epitome in what we have written. We have designed simply to give the author's *point de départ*. The second part is devoted to a special study of all the various emotions. The medical reader will be impressed with the value attached in the book to the teachings of pathology. In these special studies he will find, moreover, a remarkable presentation of the affective life—written in a most attractive style and illustrated in the happiest and most appropriate manner. For a purely psychological work, indeed, the style renders it one of the most interesting books that we have ever read. Throughout its pages there is followed a strictly scientific method—and an emotion is dissected with the skill, if not with the scalpel, of an anatomist.

JAMES HENDRIE LLOYD.

BOOKS RECEIVED.

"Analytical Cyclopædia of Practical Medicine." Chas. E. de M. Sajous, M. D. F. A. Davis Co.

"Leitfaden der physiologischen Psychologie." Ziehn. Gustav Fischer, 1898.

"Internal Medicine and Clinical Diagnosis." Jacob. W. B. Saunders, Philadelphia, 1898.

"Ueber die Tabes." P. J. Mœbius. S. Karger, 1897.

"Die Geschwülste des Nervensystems." S. Karger, 1897.

"Les Hydrocephalies." Léon d'Astros. G. Steinheil, Paris, 1898.

"International Medical Annual." E. B. Treat & Co., New York, 1898.

"An American Text of Genito-Urinary Diseases, Syphilis and Diseases of the Skin," by L. B. Bang, M. D., and W. A. Hardaway, M. D. W. B. Saunders, Philadelphia.

"The Diseases of the Stomach," by J. D. Nisbet, M. D., and William W. Van Valzah, M. D. W. B. Saunders, Philadelphia.

"Lectures on Nervous Diseases," by C. S. Elliott. A. L. Chatterton & Co., New York.

ANNUAL MEETING OF THE AMERICAN NEUROLOGICAL ASSOCIATION.

The American Neurological Association, which is to meet at the New York Academy of Medicine, No. 17 West Forty-third Street, on Thursday, Friday and Saturday, May 26, 27 and 28, 1898, announces the following preliminary programme.

"A Case with the Combined Symptoms of Myxœdema and Graves's Disease," by William Osler, M. D.

"A Contribution to the Surgery and Pathology of the Gasserian Ganglion," by W. W. Keen, M. D., and William G. Spiller, M. D.

"A Case of Syringomyelia, and Two Cases of Tabes, with Sensory Dissociation on the Trunk," by Hugh T. Patrick, M. D.

"Report of a Case of Non-traumatic Purulent Pachymeningitis, with Autopsy," by W. M. Leszynsky, M. D.

"A Case of Landry's Paralysis, with Necropsy and Microscopical Examination," by Charles K. Mills, M. D., and William Spiller, M. D.

"A Case of Alcoholic Meningitis Simulating Brain Tumor," by Theodore Diller, M. D.

"A Case of Friedreich's Ataxia Presenting Some Unusual Features," by Theodore Diller, M. D.

"On Myotonia," by George W. Jacoby, M. D.

"A Summary of the Symptoms Found in Sixty-one Cases of Locomotor Ataxia, with Additional Remarks," by W. H. Riley, M. D.

"The Prognosis and Treatment of Compression Lesions of the Cord," by Graeme M. Hammond, M. D.

"Congenital Facial Diplegia," by H. M. Thomas, M. D.

"On Scleroderma," by F. X. Dercum, M. D.

"A Case of Amyotrophic Lateral Sclerosis Presenting Bulbar Symptoms, with Necropsy and Microscopical Studies," by F. X. Dercum, M. D., and W. G. Spiller, M. D.

"A Consideration of the General and Special Clinical Aspects of Herpes Zoster," by Leonard Weber, M. D.

"The Neurological Aspect of Public School Education," by John Punton, M. D.

"Family Periodic Paralysis," by E. W. Taylor, M. D.

"The Pathological Anatomy of Amaurotic Family Idiocy," by William Hirsch, M. D.

"Some Considerations upon the Significance of the Metaplasma Granules of the Neuron," by Ira Van Gieson, M. D.

"A Case of Syphilitic Multiple Neuritis," by Frank R. Fry, M. D.

"Report of a Case of Amaurotic Family Idiocy, with Autopsy," by Frederick Peterson, M. D.

"The Pupil in Intracranial Hemorrhage," by George J. Preston, M. D.

"Report of a Case of Cerebellar Tuberculosis," by V. P. Gibney, M. D.

"A Note on the Temperature in Nervous and Mental Inefficiency," by Smith Baker, M. D.

"Results of Thyroidectomy in Eight Cases of Graves's Disease," by J. Arthur Booth, M. D.

"Presentation of a Case of Graves's Disease with Double Optic Neuritis (Choked Disks)," by J. Arthur Booth, M. D.

"Experimental Researches on the Localization of the Sympathetic Nerve in the Brain and Spinal Cord, with Contributions to the Physiology of the Sympathetic," by B. Onuf, M. D., and Joseph Collins, M. D.

"Reflections on the Nosology of the So-called Functional Diseases," by Joseph Collins, M. D., and Joseph Fraenkel, M. D.

"Note on Detecting in Perspiration the Use of Cocaine by Habitués," by Richard Dewey, M. D.

"A Case of Huntington's Chorea, with Remarks on the Propriety of Naming the Disease Dementia Choreica," by Frank K. Hallock, M. D.

"Long Remissions in Epilepsy, and Their Bearing upon Prognosis," by Wharton Sinkier, M. D.

"On Cerebral Tumor," by Alfred Wiener, M. D.

"A Case of Syringomyelia with Unusual Symptoms; Autopsy: Microscopical Report," by William N. Bullard, M. D.

"On Hysteria in Early Life," by B. Sachs, M. D.

"The Bruce Microtome," by C. Eugene Riggs, M. D.

"Pressure Myelitis Due to Tubercular Thoracic Abscess," by C. Eugene Riggs, M. D.

"Considerations on Amyotrophic Lateral Sclerosis," by Edward D. Fisher, M. D.

"Asthenic Bulbar Paralysis," by Philip Zenner, M. D.

"Regeneration of Nerve Fibres in the Central Nervous System," by W. L. Worcester, M. D.

"Chlorosis and Retino-pupillitis," by H. M. Bannister, M. D.

"A Case of Erythromelalgia, with Microscopical Examination of Tissue from an Amputated Toe," by S. Weir Mitchell, M. D., and William G. Spiller, M. D.

"Report of a Case of Tabes with Hepatic Crises and Autopsy," by William C. Krause, M. D.

"Report of a Case of Hemiatrophy of the Face," by Charles W. Burr, M. D.

THE
Journal
OF
Nervous and Mental Disease

Original Articles.

ON LANDRY'S PARALYSIS, WITH THE REPORT
OF A CASE.

BY CHAS. K. MILLS, M.D., AND WM. G. SPILLER, M.D.

From the William Pepper Laboratory of Clinical Medicine, University
of Pennsylvania.

The literature of Landry's paralysis has become so extensive that it might be supposed that the pathology of the disease is thoroughly understood, but this is far from the truth. It is hoped that the report of the following case will add something of value to our knowledge of this subject.

H. W., a well-developed man of thirty-five years, a boiler-maker, was admitted to the Philadelphia Hospital, November 26th, 1897. He denied having had syphilis, but acknowledged that he had been guilty of alcoholic excess and had had gonorrhœa.

For a month he had been feeling weak, but it was not until November 20th, 1897, that his symptoms became serious. At this time he complained of numbness in both hands, and of a slight inability to execute finer movements. He had stated previously that he had experienced numbness in both feet, but attributed this to the amount of walking he was obliged to do.

The next day, November 21st, while attempting to run for a street car, he fell, and was unable to rise without assistance. On November 22d, his legs gave way again while he was in a store, and he was unable to rise without help. On November 23d he was still able to walk on the street, but fell, and could not rise until he was picked up and assisted to his home. During November 23d and 24th the loss of power gradually increased, more markedly at first in the upper limbs, until by

November 25th he was completely paralyzed in all the extremities. He began at this time to have considerable difficulty in breathing, and also had a cough. This was his condition when he was admitted to the Philadelphia Hospital, to the service of Dr. Mills, November 26th.

When he was examined, November 27th, he was perfectly conscious, and gave intelligently a full history of his illness, but he suffered from intense dyspnoea. He spoke slowly and distinctly. He was able to move the pelvis up and down to a slight extent, and to rotate the lower limbs a little at the hip joints, but otherwise he was completely paralyzed in all four extremities. The tendon reflexes in the lower limbs were entirely lost, and in the upper the response to a tap over the biceps muscles was feeble and soon exhausted. The limbs were slightly rigid. The dysphagia was great, and he choked in drinking. The sphincters were not affected. Numerous râles were heard in the lungs. The expansion of the chest was good on both sides. The percussion revealed nothing abnormal. The pulse was rapid, but forcible. The apex beat of the heart was in the fifth interspace and was strong. No cardiac murmurs were detected.

Sensation for heat, cold, pain and touch was normal everywhere. He had complained at first of some pain in the posterior part of both thighs, and especially behind the knees. Pain had passed away at the time of examination, except in the posterior part of the cervical region, especially when the head was not supported. No tenderness was present in the back of the neck on pressure, or over any of the nerve trunks of the body or limbs, except in a small area on the right side, under the outer third of the clavicle.

The ocular muscles were not paralyzed, and the pupils responded to light and in accommodation. The ophthalmoscopic examination revealed nothing abnormal.

The patient stated that he had noticed swelling of the face in the morning, especially under the eyes, for some time. The color of the urine was reddish; its reaction was acid; its specific gravity was 1024, and it contained a little sediment, in which bladder cells and leucocytes were found, but neither albumin nor sugar was present.

The man became gradually worse, and he died November 27th, 1897.

The notes of the necropsy, made twenty-four hours after death, by Dr. William B. Jameson, are as follows:

The heart contained fluid blood and some clots; its valves were normal, and its weight was 400 grammes. At the left apex of the lung, and along its posterior surface, were extensive pleural adhesions. The left lung was markedly emphysematous, and showed extensive catarrhal pneumonia in

the lower lobe. Its weight was 1,100 grammes. The right lung showed minute patches of catarrhal pneumonia throughout. It was also emphysematous, but less so than the left. Its weight was 1,250 grammes. Some purulent excretion was noted in the bronchi of both lungs. The spleen was normal. The left kidney was distinctly red in color, and the capsule stripped easily, leaving a smooth surface. The cortex was normal in depth, and the vessels were not notably thickened. No gross lesions were observed. The weight was 180 grammes. The right kidney presented the same appearance as the left, and its weight was 190 grammes. The surface of the liver was smooth. The capsule was not thickened. Considerable hypostatic congestion and numerous, but not extensive, areas of fatty infiltration were observed. Its weight was 2,270 grammes. The meninges of the cord were normal, and no gross lesions were observed in the cord or brain. The cord appeared to be hyperæmic. The pia matter was deeply injected. The weight of the brain was 1,450 grammes.

The gross examination, therefore, showed chronic pleurisy of the left side, bilateral catarrhal pneumonia, pericardial effusion (about two ounces of clear serum), emphysema and hyperæmia of the gray matter of the cord, pons and oblongata.

One of the external popliteal nerves was examined by the method of "teasing" and staining with a one per cent. solution of osmic acid. Many of the medullary sheaths were greatly swollen, and in some fibres the myelin was entirely broken up into black balls. The nuclei were also more numerous. Many of the fibres appeared normal, or only slightly altered, and the process had evidently been of too short duration to cause involvement of all the fibres.

In one of the cutaneous nerves removed from the sole of the foot the lesions were less intense. The carmine stain showed many small axis cylinders in the external popliteal nerve, and occasionally one or more axis cylinders somewhat swollen.

The cells of the ventral horns throughout the cord were tumefied and more or less rounded; their centres were more homogeneous than normal, and contained scattered granules stained purple with the thionin after Lenhossék's method; the nucleus was displaced to the periphery of the cell, but hernia of the cell was not observed. The nucleolus stained an intense purple, and contained often one, two or three vacuoles, which are recognized as existing in normal nucleoli. At the periphery of the cells the chromophilic elements were still preserved, and the dendrites were intact. The destruction began in the chromophilic elements situated about the nucleus, and evidently had not involved all the cellular contents. Some of the cells of the ventral horns were normal, which

we should expect, in view of the fact that many nerve fibres were preserved. The carmine stain, as well as the Nissl stain, showed the tumefaction of the cells and the displacement of the nucleus.

The cells in the sacral region were as much affected as those elsewhere in the cord, notwithstanding the absence of vesical and rectal symptoms. The cells of the column of Clarke appeared to be normal.

The medullary sheaths in the anterior and posterior roots were stained black by Marchi's method, and this was noticed especially in the extramedullary portion. A slight cellular infiltration was seen within the roots, meninges and cord in some of the sections.

In the thoracic segments, in which more roots were obtained than in sections from other parts of the cord, the posterior roots presented a number of swollen axis cylinders when the carmine was employed as a stain; a few swollen ones were also found in the anterior roots, but they were not as numerous and not as large as those in the posterior roots.

Sections from the cervical, midthoracic and lumbar regions stained by the method of Marchi revealed numerous black dots scattered all over the transverse area of the cord, but these are hardly to be considered as indicative of degeneration. We have seen sections from presumably normal spinal cords stained by this method in which these same diffuse black masses were present in moderate amount. The swelling of the axis cylinders in the spinal roots, as seen by the carmine stain, renders the pathological nature probable of some at least of these black dots in the roots.

Some of the cells of the twelfth and of the motor fifth and tenth nuclei stained diffusely, but this may have been due largely to postmortem changes, for the nuclei within these cells were not displaced.

One or two prominent accumulations of round cells were found within the oblongata, but not elsewhere.

The cerebral cortex appeared to be intact, and the giant cells of the paracentral lobule were normal.

No hæmorrhages were observed anywhere within the central nervous system.

No microorganisms could be found, either by the thionin stain or by Gram's method, and cultures made from the spinal cord, and examined by Dr. Samuel S. Kneass, yielded negative results.

We understand by Landry's paralysis a disease in which the rapid loss of motor power usually begins in the lower limbs, and the paralysis is flaccid and associated with paræsthesia and loss of the tendon reflexes. The upper ex-

tremities are soon involved, and bulbar symptoms develop after a few days. Pain is not a prominent symptom. Death occurs within a week or ten days, though in some cases it may be delayed, and in others more rare recovery may ensue. There may be a descending, as well as an ascending form of paralysis. The electrical reactions are normal, and the patients are perfectly conscious of their condition.

This may be considered quite a characteristic picture of Landry's paralysis, but the lines cannot always be sharply drawn, either clinically or histologically. Thus Oppenheim¹ says that spontaneous pain is exceptional, acknowledging thereby that it may occur. He likewise says that in some cases reaction of degeneration and vesical and rectal symptoms are noted. Muscular degeneration has also been observed.

It is evident that the distinction between Landry's paralysis and polyneuritis cannot be sharply made if Oppenheim's views are accepted, and this is especially true if we follow the teaching of Nauwerck and Barth.²

Goldflam reports a case of Landry's paralysis which for a time he regarded as one of paroxysmal paralysis.³

Raymond,⁴ after an excellent presentation of the subject of acute ascending paralysis, says that the disease may ascend or descend, may begin or end with bulbar symptoms, and the electrical reactions may be altered. Disturbance of sensation may be absent, slightly pronounced, or very marked. Paræsthesia, pain, hyperæsthesia, or anæsthesia may be present. The tendon reflexes may be preserved or abolished.

Bailey and Ewing⁵ have tabulated a large number of cases of Landry's paralysis, and much labor will be spared

¹ Oppenheim: *Lehrbuch der Nervenkrankheiten*, p. 357.

² Nauwerck and Barth: *Ziegler's Beiträge*, vol. v.

³ Goldflam: *Deutsche Zeitschrift f. Nervenheilkunde*, vol. xi., Nos. 3, 4.

⁴ Raymond: *Leçons sur les Maladies du Système Nerveux*, Deuxième Série.

⁵ Bailey and Ewing: *The New York Medical Jour.*, 1896, No. 2.

future writers on this subject. They state that the nerves were examined in osmic acid only in two of the sixteen cases without lesions, and in none of these cases were the most improved methods for staining the cells employed. It matters little how skilled an investigator may be, he will be unable to detect very early cellular changes by the carmine stain, or early nerve lesions by the hæmatoxylin stain of Weigert. The most reliable method for the determination of beginning alteration in nerve fibres is the method of "teasing" and staining the tissue in the fresh state with osmic acid.

Bailey and Ewing state that in nine of the fourteen cases in which changes were found in the cord alone, the nerves were not examined, and in only one of these nine cases were the clinical symptoms of neuritis present. This latter statement is of limited value. It is the experience of every oculist that optic neuritis may be present and cause little functional disturbance, or that impairment of sight may be noticed with clear media and apparently normal fundus. Organic changes in the body may be latent for some time. The symptoms of neuritis may be chiefly those of involvement of motor fibres, as in this case we report.

Bailey and Ewing believe that the remaining five cases conclusively demonstrate that a lesion limited to the cerebrospinal axis may produce acute ascending paralysis. These five cases, we take it, are those of Eisenlohr, Hoffmann, Immermann, Hlava, and Cettinger and Marinesco. Unless the nerves were examined by osmic acid, these cases are not conclusive of the exclusively spinal origin of Landry's paralysis.

In regard to Eisenlohr's⁶ case, we find the statement that transverse sections of the anterior roots from different parts of the cervical region, stained with carmine, presented normal fibres. We find no mention of a careful

⁶ Eisenlohr: *Virchow's Archiv*, vol. lxxiii., p. 73.

examination of the peripheral nerves. This case, therefore, is not conclusive.

In Hoffmann's⁷ case pathological changes were only found in the facial nerve; all the other nerves examined were normal. The intramedullary portions of the anterior roots were much altered. The nerves had been hardened in Müller's fluid, and do not seem to have been examined in the fresh state. We are sure that early lesions of nerve fibres may be overlooked in preparations hardened in Müller's fluid.

In regard to Immermann's⁸ case, the muscles and peripheral nerves were intact. No mention is made of the method of examination of the nerves. Immermann says that the case shows that there is a form of acute anterior poliomyelitis which clinically corresponds to Landry's paralysis.

In regard to Hlava's⁹ case, the sciatic and ulnar nerves were examined, and while mast cells were found in both, no degeneration was detected. Unfortunately no mention of the method of examination of the nerves is given in this abstract, and the original paper is not accessible to us.

In Cettinger and Marinesco's¹⁰ case the alterations in the central nervous system were great, without appreciable lesions in the peripheral nerves.

Of the six cases of acute anterior poliomyelitis which Bailey and Ewing would include as cases of Landry's paralysis, they state that four show an involvement of the cord alone. In the abstract of Rissler's¹¹ cases we find that secondary degeneration of motor nerves was observed in the acute cases, and that in the more chronic ones the roots were diminished in size.

In Redlich's case degeneration was noted in the brachial plexus.

⁷ Hoffmann: *Archiv f. Psychiatric*, vol. xv., 1884.

⁸ Immermann: *Neurologisches Centralblatt*, 1885, p. 304.

⁹ Hlava: *Schmidt's Jahrbücher*, 1891, 232, p. 244.

¹⁰ Cettinger and Marinesco: *Semaine Médicale*, 1895, p. 45.

¹¹ Rissler: *Neurologisches Centralblatt*, 1889, p. 422.

In Goldscheider's the nerves were not examined.

In Dauber's²¹ case of poliomyelitis it is distinctly stated that the anterior roots were pathologically altered.

In regard to the cases collected by Bailey and Ewing, in which the peripheral nerves alone were affected, these writers state that only one case shows that a lesion limited to the peripheral nerves may cause typical symptoms of fatal acute ascending paralysis. This is evidently Dejerine's case, which was examined before Nissl's stain was in vogue.

In Eisenlohr's¹³ case (Case 1) the central nervous system was put in Müller's fluid, and Nissl's stain was probably not employed.

A total of twenty-eight recorded cases of Landry's paralysis in which lesions were found, as collected by these writers, must make us somewhat sceptical toward the report of those cases in which no lesions were noted. Only in a few cases have bacteria actually been observed.

In the case of Landry's paralysis reported by Bailey and Ewing (l. c.) intense perivascular cellular infiltration was found within the cord, which we have not noticed in our case, except in one or two regions of the oblongata.

The changes which they found in the motor cells are not unlike those described by us. The peripheral nerves were not examined by them, which is much to be regretted. The fact that the nerve roots in the upper thoracic and cervical segments were intact, especially as considerable cellular infiltration was seen within them, does not exclude the possibility of peripheral degeneration. It is very true that, if such degeneration had been present, it might have been secondary to the cord lesions; but it might also have occurred at the same time as the spinal lesions. A toxin may affect the most distal portion of the peripheral neuron simultaneously with its attack on the cord. We question the possibility of deciding from the

²¹ Dauber: *Deutsche Zeitschr. f. Nervenheilk.*, vol. iv.

¹³ Eisenlohr: *Deutsche med. Wochenschrift*, No. 38, 1890.

nature of the cellular lesions whether the change is primarily within the cell or in the terminal ramifications of the neuron.

In Hun's¹⁴ case, which, clinically, was quite a typical one of Landry's paralysis, a slight cerebral and spinal meningitis of quite recent origin, a degeneration of some of the fibres of the anterior roots of the cauda equina, and a thickening and infiltration of the walls of the anterior spinal vein were noted. Some portions of the cord were hardened in alcohol, but no detailed statements are given of the condition of the motor cells as examined by Nissl's stain. No changes were found in the peripheral nerves by osmic acid.

In this case the moderate infiltration of the spinal pia and its vessels with small round cells might be regarded as the early stages of meningitis, and, had the patient lived longer, it is possible that meningitic pains would have been experienced. We may thus understand how easily sensory symptoms are added to the purely motor ones of Landry's paralysis.

Hun believes that his case goes far to prove that acute ascending paralysis may exist without any appreciable lesion. In view of this statement, we cannot help regretting that more exact information of the condition of the cells, as stained by Nissl's method, could not be given.

Dejerine¹⁵ states that certain toxins cause paralysis without neuritis histologically appreciable, and from the text it is evident he has not in mind myelitis as the cause of such paralysis. He refers to the experiments of Charrin as a proof of his statement. Babinski and Charrin¹⁶ produced motor paralysis in rabbits by the injection of the bacillus pyocyaneus, or merely the soluble products of this microorganism obtained by cultures. They ex-

¹⁴ Hun: *The New York Medical Journal*, vol. liii., p. 609.

¹⁵ Dejerine: *La Médecine Moderne*, Dec. 21, 1895.

¹⁶ Babinski and Charrin: *Comptes rendus de la Société de Biologie*, 1888, p. 257.

amined the muscles, nerves and central nervous system of these animals at different periods of the paralysis, even after two months had elapsed, and employed various methods, but their results were absolutely negative. These experiments show us that we may expect negative findings in some cases of Landry's paralysis.

Hun (1. c.) states that in every reported case of Landry's paralysis due to neuritis few or no symptoms of bulbar paralysis were observed, and that these cases also presented decided disturbance of sensibility, manifested by severe pain or by extensive anæsthesia, or paralysis of the sphincters, or atrophy of the muscles, with more or less well-marked electrical reaction of degeneration, and tenderness on pressure.

In our case, in which polyneuritis was certainly present, although equally marked changes were found in the cell bodies within the cord, bulbar symptoms were manifested in great dyspnœa and dysphagia. No disturbances of objective sensation were present, and only slight pain, if we include the pain experienced for a short time and limited to one or two regions. Paræsthesia was noted, but no involvement of the sphincters, no muscular atrophy, and no tenderness on pressure, except in one very limited area. Unfortunately the electrical reactions were not taken. This absence of tenderness of the muscles on pressure is interesting, as a number of writers state that even in the motor form of neuritis in which sensation is apparently normal, pressure of the affected muscles usually causes pain.

The case reported by Ballet and Dutil¹⁷ may possibly be regarded as representing a later stage of a condition similar to that in Hun's case. Sensation in all its forms was intact. The spinal vessels, especially those in the anterior horns, were enormously dilated, and considerable leucocytic infiltration was found. The cells of the posterior

¹⁷ Ballet and Dutil: *Bulletins et Mémoires de la Société Médicale des Hopitaux de Paris*, 1895, p. 684.

horns, those of the columns of Clarke, and especially those of the anterior horns, presented destruction of the chromophilic elements, cloudiness of the cell protoplasm, destruction or rupture of the cellular processes, and nuclear changes. It was, therefore, a case of diffuse myelitis. The roots and peripheral nerves showed an early stage of degeneration. No microbes could be found.

The case reported by Marie and Marinesco¹⁸ also shows that ascending paralysis, especially in the wider acceptance of the term, may be due to disease of the central nervous system, although Nauwerck and Barth stated, at the time they wrote their paper, that the proof of such an origin of the disease had not been given.

This case reported by Marie and Marinesco was quite a typical one of Landry's paralysis, except that the patient suffered intense pain in the entire body. Objective sensation was diminished only for a time. Intense mononuclear leucocytic infiltration was observed in the anterior horns as well as infiltration of a less intense degree in the posterior. Some of the ganglion cells of the anterior horns were swollen and the chromophilic elements had disappeared. Some of the processes and cell bodies were divided. In the cervical region where the disease was not so far advanced, it was easy to see that the elements nearest the nucleus had disappeared, while those at the periphery of the cell were preserved. The nucleus was displaced. The peripheral nerves seemed to be intact.

We have mentioned this case somewhat in detail on account of its importance. It presents cellular changes from myelitis, some of which are very much like those seen when the axis cylinder is cut, and it shows the difficulty in deciding as to the primary or secondary nature of the cellular changes. It proves that the peripheral nerves are not always diseased when the cell body is much altered;

¹⁸ Marie and Marinesco: *Bulletins et Mémoires de la Société Médicale des Hopitaux de Paris*, 1895, p. 659.

and it shows, finally, that Landry's paralysis may be due to purely central lesions.

The central form of Landry's paralysis, according to Bruns,¹⁹ is chiefly in the area of the cord nourished by the central arteries. This central form of Landry's paralysis, like the acute anterior poliomyelitis, may be due to a poison which gains entrance to the cord chiefly through the anterior spinal arteries, which we know nourish the gray matter. It is not necessary that the bacteria themselves should be found within the cord, as numerous experiments have shown that the toxins alone are sufficient to produce such changes. Ballet,²⁰ Phisalix and Charrin,²¹ refer to a number of these investigations.

The case reported by Göttinger and Marinesco²² was due to central lesions also, and the peripheral nerves did not present any appreciable alteration. Sensation for touch, pain and temperature was much diminished. Bacteria were numerous within the cord. Marie in discussing this case (l. c.) says that certain authors have been wrong in stating that acute ascending paralysis is due to peripheral neuritis, but, "in reality, it is due, purely and simply, to lesions of the nervous centres." Our case would hardly support this statement.

The close connection of Landry's paralysis and typical polyneuritis may be seen in a case reported by Raymond.²³ The paralysis was not of the ascending type, but he states that no clinician would have hesitated at first to call the case one of Landry's paralysis, although the later developments of the case made this diagnosis untenable. The muscles and nerves of the lower extremities became very

¹⁹ Bruns: *Allgemeine Zeitschrift für Psychiatrie*, vol. liii.

²⁰ Ballet: *Psychoses et Affections Nerveuses*.

²¹ Phisalix and Charrin: *Comptes rendus de la Société de Biologie*, No. 3, 1898.

²² Göttinger and Marinesco: *Bulletins et Mémoires de la Société Médicale des Hôpitaux de Paris*, 1895, p. 63.

²³ Raymond: *Leçons sur les Maladies du Système Nerveux, Deuxième Série*.

painful on pressure, the muscles of the extremities atrophied, and presented the reaction of degeneration.

The changes which occur within a nerve in parenchymatous inflammation are like those of descending degeneration and, notwithstanding the many causes of peripheral neuritis, the lesions of most forms are similar, though possibly the perfection of our methods of examination may reveal a variety of histological forms. The changes in a nerve undergoing degeneration consist of multiplication of the nuclei of the sheath of Schwann, increase in the amount of protoplasm within the fibre, destruction of the myelin with the formation of balls of various sizes, and destruction of the axis cylinder. When the neuritis is due to traumatism, the inflammation is said to be chiefly in the connective tissues and vessels of the nerves, and the parenchyma is only secondarily affected.

Babinski voices the opinion of many neuropathologists when he says that in peripheral neuritis—as distinguished from central or secondary neuritis in which the nerves are altered secondarily to lesions within the spinal cord—the nerves are only altered at their periphery, and their trophic centres appear perfectly normal, or, at least, are only slightly altered.

It is exceedingly difficult when the neuritis is supposed to be due to a toxic or infectious agent, to decide whether the cell of origin is altered primarily or secondarily. Experimentally it has been shown that very decided changes occur within the cell when its fibre has been cut (J. Babinski,²⁴ p. 662). Ballet²⁵ discusses the subject quite fully. He says *périphéristes* believe that the nerve may be altered independently of the cell body, i. e., the peripheral part of the neuron is to a certain extent more susceptible to morbid processes than the corpus. He cites as supporters of this view Leyden, Dejerine, Pitres and

²⁴ Babinski: *Traité de Médecine*, vol. vi.

²⁵ Ballet: *Psychoses et Affections Nerveuses*.

Vaillard, and Strümpell. These authors support their opinion on the fact that more or less pronounced lesions of the peripheral nerves may be found with few changes in the roots, and none in the cord.

The centralistes, on the other hand, believe that the cell is also involved, though perhaps only functionally. Among these may be mentioned Erb, Remak, Eisenlohr, Charcot, Brissaud, Babinski and Marie. These base their opinion on the bilaterality and symmetry of the nerve lesions; on the topography of the sensory or motor disturbances which does not always correspond to the distribution of the nerve trunks, as seen, for example, in the escape of the supinator longus muscle in lead palsy; and on the degenerative rather than the inflammatory character of the nerve lesions (Erb). Babinski and Ballet state that it is quite possible to have a truly inflammatory neuritis localized to a limited area, and that the changes within the nerve below this point are of a degenerative character.

Ballet (l. c. page 359) reports a case which shows in some of the features a resemblance to Landry's paralysis, though of long duration. A man became weak in the lower limbs from unknown cause, and had numbness and tingling. Within a few weeks the weakness became paralysis and atrophy was noticed. The upper limbs also became involved, and reaction of degeneration was noted. With the exception of numbness and tingling the sensation was normal. The tendon reflexes were absent, and the sphincters were intact. The peripheral nerves were found much degenerated, and the anterior roots were only slightly affected. In the anterior horns of the cord the cells were less numerous than normal; they were swollen, and the nucleus was displaced toward the periphery. The symptoms had lasted eight months.

The cellular changes which Ballet found in this case (l. c., p. 365) were very much like those we describe. He speaks of the destruction of the chromophilic elements, at first of those about the nucleus, and finally of all within

the cell; and of displacement and change of form and more intense staining of the nucleus. The vessels and neuroglia of the cord were normal.

Ballet (l. c.) after referring to the experimental work of Nissl, B. Onufrowicz, and Marinesco, reports cellular changes very similar to those in his case of neuritis; changes which were found by Dutil in guinea pigs after section of the sciatic nerve. Only in one of the three animals was degeneration of the central end of the cut nerve found, and this animal was killed thirty-seven days after the operation. Some of the cells of the anterior horn had disappeared. This degeneration of the central end of the cut nerve was not found when the guinea pigs were killed six days or seventeen days after the operation. Ballet believes that this degeneration was secondary to the cellular changes, and was not an ascending degeneration. He states that the cases of neuritis in which the cells of origin within the cord have been found normal have not been studied by Nissl's method, though in some cases the neuritis may not be sufficiently intense to cause cellular alteration.

Marinesco also has explained the so-called ascending degeneration of the central end of a cut nerve on the supposition that it is secondary to changes within the cell body, and is really of the nature of Wallerian degeneration.

Marinesco says that Cettinger, Korsakoff, Köpper, Schaffer, Erlitzky, Achard and Soupault have described lesions of the cells of the anterior horns in alcoholic paralysis; Dejerine and Certeau in diphtheria; Oppenheim, Popoff, Rosenbach in lead palsy; and that Fuchs, Goldscheider and Moxter, Giese and Pagenstecher, Ballet and Dutil have found these cellular changes in polyneuritis. Much evidence, therefore, is in favor of the view that poisons have a very decided effect on the motor cells of the anterior horns.

Marinesco²⁶ describes a case of polyneuritis in which

²⁶ Marinesco: *Revue Neurologique*, 1896, p. 129.

the nerves were found greatly altered; the anterior and posterior roots seemed to be intact, but changes were found within the cell body very much like those in the case we report. He speaks of a resemblance of these cellular changes to those which occur when a nerve is cut, and states that he is the first to call attention to this. Figure 17 of his paper would represent very well one of the cells of the anterior horns in our own case. He calls attention to the fact that Strümpell and Raymond have believed that a toxic agent may act at the same time on the nervous centres and on the peripheral nerves. He believes that he can make a distinction between the secondary changes in a nerve cell, as seen when its fibre is cut, and primary changes, such as occur after compression of the abdominal aorta. In the latter the destruction of the chromophilic elements begins at the periphery of the cell, the perinuclear layer is intact, and the nucleus is central, while in later stages the trophoplasm (the achromatic portion) of the cell presents large spaces resulting from its partial destruction.

Marinesco regards this degeneration of the trophoplasm as an early lesion in all acute primary affections of the spinal cord, and has observed it in two cases of Landry's paralysis. Whenever the trophoplasm is altered, the lesion is irreparable, for there is no regeneration of nerve cells as shown by the work of Stroebe.²⁷

The question, therefore, as to the primary or secondary involvement of the cell of origin of the peripheral neuron is one of some importance. Restoration of function is not uncommonly noted when the lesion begins in the axis cylinder and the cell is altered secondarily, but according to Dejerine,²⁸ not a single case of diffuse myelitis or of poliomyelitis with restoration *ad integrum* has been reported.

Disease of the cell of origin of the motor nerve fibre

²⁷ Stroebe: *Centralblatt f. allgemeine Pathologie und pathologische Anatomie*, vol. vi., 1895.

²⁸ Dejerine: *La Médecine Moderne*, Dec. 21, 1895.

has been found by other investigators in neuritis. Dejerine and Theohari²⁹ have found it in neuritis of the seventh nerve, and Soukhanoff³⁰ recently in polyneuritis. Other cases are on record.

Schaffer³¹ has studied the cells in the anterior horns in several cases of tabes in which trophic lesions were observed, and has found that the cellular changes are the same whether there is amyotrophy, osteopathy or arthropathy. In the early stages the chromophilic elements about the nucleus are broken up into granules, and only when the cell body is affected, in toto, are the nucleus and nucleolus altered. The dendrites are involved later than the cell body, and may be found relatively intact when the cell body is seriously altered. The partially degenerated cells are more numerous than the totally degenerated. Schaffer concludes from his studies of cellular lesions from, toxic, infectious and trophic causes, as well as from direct disturbance of nutrition, that the Nissl stain does not show specific changes in any case, and that in every form of cellular disease the lesion is chromatolysis, which in its essential features is always the same. This chromatolysis Schaffer does not regard as a *lésion banale* in the sense used by Dejerine and Thomas, but looks upon it as an index of the cell's vitality.

Mourek and Hess,³² after performing a number of experiments in poisoning rabbits, concluded that in all the cases the chromatolysis was the same, and that the Nissl elements presented no characteristic alterations for the different poisons. The investigations of Van Gieson,³³

²⁹ Dejerine and Theohari: *Comptes rendus de la Société de Biologie*, Dec. 4, 1897.

³⁰ Soukhanoff: *Abstract in Revue Neurologique*, No. 2, 1898.

³¹ Schaffer: *Monatsschrift f. Psychiatrie und Neurologie*, vol. iii., No. 1.

³² Mourek and Hess: *Revue Neurologique*, 1897, p. 667.

³³ Van Gieson: *Medical Record*, April 9, 1898, p. 526. Ewing: *Idem*.

and more recently those of Ewing, show us that the importance of chromatolysis is not held to be equally great by all histologists.

The early changes which Schaffer has found in the spinal cells of tabetic patients are about the same as those observed by us in the case of Landry's paralysis, and like those noted in cases reported as polyneuritis. Schaffer believes that the tabetic changes are primarily in the cell body, but we must confess that we are not convinced of the correctness of this view, and it is possible that such cellular alterations in tabes are secondary to neuritis. In this sense Dejerine³⁴ would still be right in that he believes the muscular atrophy of tabes is due to neuritis. Schaffer found that normal cells were mingled with diseased ones. This could be explained by the fact that usually in neuritis some of the nerve fibres are intact, and secondary changes in the cells are only found in those belonging to the altered fibres; it could, however, be used to explain the partial involvement of nerve fibres if the cellular changes are regarded as primary. Schaffer believes that the partial degeneration of a nerve cell causes degeneration at the periphery of the neuron, in the terminal ramifications of the fibre, and as the cellular changes increase the degeneration of the fibre creeps upward. May it not be equally true that the lesion is primarily in the nerve termination, or simultaneously in the nerve termination and in the cell body? May it not be true that the peripheral lesion leads to partial degeneration of the cell body? It may be that the portion of the neuron furthest from the nucleus, as Erb and Strümpell believe, is the portion most susceptible to toxic agents. We acknowledge the force of all that Schaffer says, but we cannot regard his arguments as final. It seems to us that the question as to the primary or secondary involvement of the motor cells in tabes and polyneuritis is still open.

³⁴ Dejerine: *Sur l'Atrophie Musculaire des Ataxiques*.

Berger³⁵ has found the cells of the anterior horns much degenerated in cases of dementia paralytica, and among other changes he describes conditions very similar to those seen by us in our case of Landry's paralysis. He ascribes the muscular atrophy, seen at times in general paralysis, to these cellular lesions.

While cellular lesions have been noted in a number of cases of polyneuritis, they do not always occur. In at least two cases reported in the literature they were absent; these are the case of Soukhanoff³⁶ and the one of Dejerine and Thomas.³⁷ In the latter very notable lesions of the cutaneous and muscular nerves were observed, but the spinal cells examined by the method of Nissl were normal. Dejerine and Thomas think that it is possible that the nerve cells had been altered, but had recovered, inasmuch as the neuritis had improved. They think also that chromatolysis of the cells, observed in intoxications and infections, is interesting cytologically, but has little real importance, and may not cause any symptoms.

The changes in the cells of the anterior horns which Ballet and Lebon have found in myelitis produced in the cords of rabbits by the injection of cultures of the pneumococcus and the staphylococcus (Ballet, l. c. 434) consist in destruction of the chromophilic elements, and no mention is made of the peripheral zone of degeneration in the cell seen in compression of the abdominal aorta. In Ballet and Lebon's experiments the roots and peripheral nerves were intact, or only slightly altered. The cells must have been primarily altered, and the changes were not unlike those observed after section of the nerve fibres.

Ballet and Dutil,³⁸ in a paper read at the recent con-

³⁵ Berger: *Monatsschrift f. Psychiatrie und Neurologie*, vol. iii., No. 1.

³⁶ Soukhanoff: *Archiv de Neurologie*, 1896, vol. i., p. 177.

³⁷ Dejerine and Thomas: *Comptes rendus de la Société de Biologie*, 1897.

³⁸ Ballet and Dutil: *Monatsschrift f. Psychiatrie und Neurologie*, vol. ii., No. 5, p. 397.

gress in Moscow, stated that different poisons affect the cells differently, but that the early changes caused by compression of the abdominal aorta are much like the secondary cellular changes caused by cutting a peripheral nerve, and that, while the distinction of primary and secondary cellular changes is proper, under certain circumstances, it is impossible.

It seems most probable that the nucleus is a very important part of the cell, and that on the normal condition of this the health of the entire neuron depends, but it is not unreasonable to believe that the axis cylinder is, to some extent, perhaps only a limited extent, independent of the cell body. It is very difficult to believe that the whole trophic function of an axis cylinder, extending, for example, from the lumbar region to the foot, can reside in a body so small as the nucleus. It has been thought that the medullary sheath is for the purposes of insulation and protection to the neuraxon, and that its interruption at the nodes of Ranvier is for the purpose of affording nutritious fluids an entrance to the axis cylinder. This seems to be a reasonable supposition.

The swelling of a few of the axis cylinders of the posterior roots in our case, as shown by the carmine stain, is interesting. The method of Marchi shows many black dots in both the anterior and the posterior roots. Changes in the posterior roots, after amputation, have been observed by a number of writers, and, therefore, we should not be surprised to observe this in polyneuritis. Fleming³⁹ has noticed very decided changes in the cells of the spinal ganglia after section of the sciatic nerve, and the degeneration of these cells was noticed at a much earlier period than that of the multipolar cells in the cord, beginning as early as the fourth day, and certainly by the seventh. Redlich, as well as Darkschewitsch (cited by

³⁹ Fleming: *The Edinburgh Medical Journal*, New Series, vol. i., 1897.

Flatau), found degeneration of the posterior roots after amputation, indicating that the process had extended even beyond the spinal ganglia.

Flatau⁴⁰ was able to observe degeneration of the posterior roots after recent amputation in man when he employed the method of Marchi.

Lugaro⁴¹ found that the cells of the spinal ganglia belonging to the sciatic nerve were much altered when this nerve in the dog was cut, but that no distinct changes in the ganglia were present when the posterior roots or the posterior columns of the cord were cut. Lugaro concluded that the cells of the spinal ganglion are altered in lesions of their peripheral branches, but not in lesions of their central. From these observations the central branch of the peripheral sensory neuron would seem to be a less vital portion of the cell than the peripheral branch.

Van Gehuchten⁴² has observed the "reaction at distance" in the cells of the spinal ganglion after division of the peripheral nerve. Schaffer,⁴³ however, was unable to find any important change in these cells in tabes when he employed the method of Nissl, although he found the posterior roots greatly degenerated.

It would seem from these various statements that the "reaction at distance" does not occur in the spinal ganglion cell when its central process is cut or diseased, and does occur after lesion of the peripheral process.

Nissl stated that most of the cells in the nucleus of the seventh nerve recover after division of this nerve. Marinesco found that after he had divided the hypoglossal nerve in rabbits the nuclei in the cells of this nerve were only a little eccentric on the twenty-fourth day after the operation. It would seem, therefore, that the return of

⁴⁰ Flatau: *Deutsche med. Wochenschrift*, No. 18, 1897, p. 278.

⁴¹ Lugaro (cited by Flatau): *Fortschritte der Medicin*, 1897, No.

15. ⁴² Van Gehuchten: *Journal de Neurologie et d'Hypnologie*, 1897, p. 279.

⁴³ Schaffer: *Neurologisches Centralblatt*, No. 1, 1898.

the nucleus to a central position within the cell is an early sign of cellular regeneration. In some cells he found the reparative process perinuclear, i. e., in the part which is first altered after division of a nerve fibre, but this was not seen in all the cells. Marinesco's⁴⁴ experiments cannot be used to determine the condition of the cells when peripheral reunion of the cut ends of the nerve fibre is prevented. Van Gehuchten⁴⁵ also has studied the cellular regeneration with similar results after experimental division of the nerve.

We have the testimony of a number of writers that "reaction at distance" occurs in both sensory and motor neurons. In addition to the quotations already made, we may state that Sano⁴⁶ found chromatolysis of the cells of the column of Clarke after amputation of the lower limb, which he explained as "reaction at distance" from injury by meningitis to the nerve processes of these cells. He also found this reaction in the motor cells of the cord. Barker⁴⁷ gives the same explanation for the chromatolysis which he found in cells of Clarke's column in two cases of epidemic cerebrospinal meningitis. The direct cerebellar tract was injured. The changes in the motor cells he explains as the result of compression of their axis cylinders by the meningeal inflammation.

This, however, is not the only explanation which could be offered for the chromatolysis in the cells of Clarke's column in such cases. Marinesco⁴⁸ has found such changes in these cells in general paralysis and in tabes, diseases in which degeneration of the posterior roots is common, and he regarded these as secondary, and like those seen in poly-

⁴⁴ Marinesco: *Comptes rendus de la Société de Biologie*, 1896, p. 930.

⁴⁵ Van Gehuchten: *Journal de Neurologie et d'Hypnologie*, 1897, p. 279.

⁴⁶ Sano: *Journal de Neurologie et d'Hypnologie*, 1897.

⁴⁷ Barker: *Brit. Med. Jour.* 1897, vol. ii., p. 1,839.

⁴⁸ Marinesco: *Revue Neurologique*, 1896, p. 633.

neuritis. Van Gehuchten⁴⁹ also has found changes in the acoustic nucleus of the oblongata after division of the eighth nerve, i. e., in the cell body of the second neuron after injury of the peripheral neuron. The chromatolysis in the cells of the column of Clarke may, therefore, be the result of injury of posterior root fibres.

Barker⁵⁰ acknowledges the possibility of the correctness of this explanation. He states that in his cases of epidemic cerebrospinal meningitis he found two distinct types of cellular change. In the first the disintegration of the Nissl bodies was especially marked in the dendrites and along the periphery of the cell, but these changes were slight, owing, he thinks, to the fact that cerebrospinal meningitis is an affection not associated with a severe toxæmia. In the second type the changes in the chromophilic elements were those described as "reaction at distance," i. e., they were chiefly in the centre of the cell and the nucleus was displaced. The first type Barker believes is the result of the direct action of the poison upon the cell body, and the second is due to injury of the axis cylinder.

He does not believe that the involvement of the posterior roots in his cases was sufficient to cut off enough of the sensory impulses to account for the changes found in almost every one of the cells of Clarke's column. This is a forcible argument, but we cannot forget the experiments of Babinski and Charrin, referred to above, which have shown that there may be a total loss of function of a nerve fibre without detectable lesions.

It is not extraordinary, therefore, that degeneration of the posterior roots should occur from polyneuritis. The involvement of the posterior roots and posterior columns in the disease has been noted by a number of observers, and

⁴⁹ Van Gehuchten: *Centralblatt f. Nervenheilkunde und Psychiatrie*, Beiheft, October, 1897, p. 15.

⁵⁰ Barker: *The Johns Hopkins Hospital Bulletin*, February, 1898, p. 33.

Redlich⁵¹ mentions such cases in his critical digest on diseases of the posterior columns of the cord. Pal and Redlich do not believe that the degeneration of the posterior roots is secondary to the neuritis. As Redlich expresses it, it is presumable that the poison acts on the entire sensory neuron at the same time. The possibility of this must, of course, be considered, but the observation of Flatau quoted above would indicate that the involvement of the posterior roots in polyneuritis may be secondary; for no other interpretation could well be given of his findings in these roots after amputation.

We do not always find the relations existing between disease of the motor cells, of the roots and of the peripheral nerves that we should expect. Thus, Gombault noted that the anterior roots were normal in a case of amyotrophic lateral sclerosis, although many of the motor cells had disappeared. Dreschfeld found marked lesions in the anterior cells of the cord while the intramuscular fibres were normal, and the nerves only slightly altered. Oppenheim reported similar conditions. V. Monakow, Zunker, Celler, Kronthal and Darkschewitsch (cited by Babinski⁵²) have noted this want of correspondence in the intensity of the cellular changes and those of the nerves and roots, and recently Luce⁵³ has reported a case in which the cells of the anterior horns were much degenerated; and the anterior roots were only slightly affected. These cases would seem to indicate that the nerves enjoy an existence independent, to a certain extent, of the cell body.

Barbacci and Campacci⁵⁴ found that the nerve cells in the rabbit undergo postmortem changes. They noticed that the chromophilic elements were paler and more indistinct than normal, and that they were changed into gran-

⁵¹ Redlich: *Centralblatt f. allgemeine Pathologie und pathologische Anatomie*, 1896.

⁵² Babinski: *Traité de Médecine*, vol. vi., p. 650.

⁵³ Luce: *Deutsche Zeitschrift f. Nervenheilkunde*, vol. xii., No. 1.

⁵⁴ Barbacci and Campacci: *Abstract in Neurologisches Centralblatt*, 1897, p. 1,042.

ules, especially about the nucleus, while the periphery of the cell was intact. In later stages vacuoles were found. Changes were seen also in the nucleus and nucleolus.

Such findings must, of course, make us cautious in accepting all the cellular changes which are described as pathological, but they can hardly destroy the value of the work of many investigators. We, as others have done, have examined the spinal cord removed from persons some hours after death, who had not suffered from spinal or peripheral nerve lesions, and have not found any such changes as are present in our case of Landry's paralysis.

Held⁵⁵ has shown that the chromophilic elements do not exist in the living cell; that they are precipitated by the addition of fluids, and that they vary in form with the fluid used. He has likewise demonstrated that vacuoles in the protoplasm are postmortem changes, and may be produced by the addition of water.

These experiments throw light on the changes which occur within the cell under the influence of poisons. If the composition of the cellular protoplasm is chemically altered by toxic substances, the postmortem precipitation must be different from that in normal cells; or, if the function of the cell is destroyed by the cutting of its axis cylinder, the composition of the cellular protoplasm is most probably changed, and the precipitation must also be different. It matters little whether or not the Nissl elements are postmortem formations, as they always present the same appearance in normal cells; they afford, when altered, an index of the diseased condition of the cell.

Some quite recent experiments by Goldscheider and Flatau⁵⁶ have shown that great alteration of the chromophilic elements may exist without disturbance of function, and in a case of pneumonia Dejerine found the spinal cells swollen and without chromophilic elements, and the cellu-

⁵⁵ Held: *Archiv f. Anatomie und Entwicklungsgeschichte*, 1895, p. 396.

⁵⁶ Goldscheider and Flatau: *Fortschritte der Medicin*, No. 7, 1897.

lar processes altered. The patient had had no motor or sensory symptoms. This case Dejerine⁵⁷ brings forward in support of his views regarding the unimportance of chromatolysis.

Ballet and Dutil⁵⁸ likewise have stated that the paralysis caused by compression of the abdominal aorta may disappear and motion be perfectly normal at a time when the spinal cells are still greatly altered.

Our conclusions, therefore, from a study of our case and of the literature, are:

1. That there is a form of ascending, flaccid paralysis, with little disturbance of sensation, with normal electrical reactions, and without involvement of the sphincters, and that this is of rapid course, usually terminating in death.

2. Other cases differ from this type by one or more atypical signs, and transitional forms occur which make the diagnosis between Landry's paralysis, polyneuritis and myelitis difficult.

3. It is possible that in some cases no lesions exist; but many of the reports of such cases date from a time when the methods of examination were very imperfect; or it may be that in these cases the lesions are in an early stage of development, the patient succumbing to toxæmia before demonstrable changes in the nervous system take place.

4. That Landry's paralysis may be due to myelitis alone.

5. In Landry's paralysis polyneuritis may be present, but changes in the cell bodies of the anterior horns will also usually be found in such cases by Nissl's stain, and it is sometimes difficult to say whether the cellular changes are primary or secondary.

6. It is probable, in some cases at least, that the entire peripheral motor neuron is attacked at the same time by the poison of the disease.

⁵⁷ Dejerine: *Comptes rendus de la Société de Biologie*, 1897

⁵⁸ Ballet and Dutil: *Monatsschrift f. Psychiatrie und Neurologie*, vol. ii., No. 5.



FIG. I.

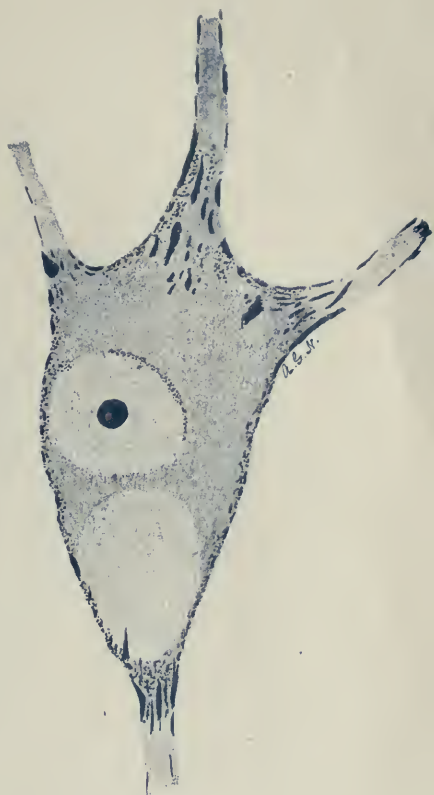


FIG. III.

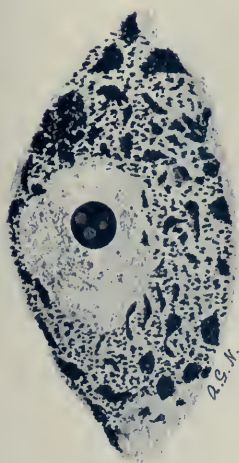


FIG. II.

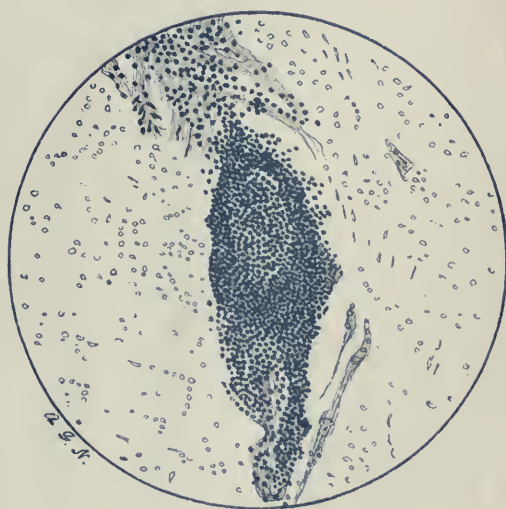


FIG. IV.



FIG. V.

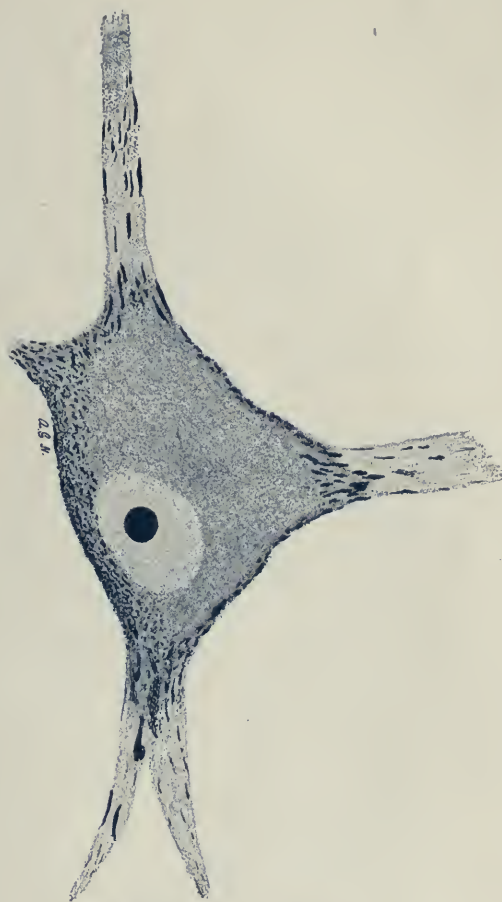


FIG. VI.



FIG. VII.

LEGENDS.

Figure 1: Normal cell from the hypoglossal nucleus.

Figure 2: Normal cell from Clarke's column.

Figure 3: Degenerated cell from the hypoglossal nucleus.

Figure 4: Round-cell infiltration in the oblongata.

Figures 5 and 6: Cells from the anterior horns of the spinal cord, showing central chromatolysis.

Figure 7: Nerve fibres from the external popliteal nerve, showing tumefaction and disintegration of the myelin.

132. *TABES UND MULTIPLE SKLEROSE IN IHREN BEZIEHUNGEN ZUM TRAUMA.* (Tabes and Multiple Sclerosis in their Relation to Trauma). E. Mendel (*Deutsche med. Wochenschr.*, p. 97, 1897).

Mendel comes to the conclusion, from his own experience, that trauma has not been clearly shown to be a cause of tabes. It often does cause a rapid development of pre-existing tabes, and in some cases tabetic patients, who until the occurrence of the trauma had been able to work, have become unable to earn their support after an accident. It is probable that the conditions connected with the trauma affect the disease, especially the long rest in bed. It is a well-known fact that tabetic patients have very imperfect use of their legs after a long confinement to bed. Hygienic conditions after such accidents also play a part in the development of tabetic symptoms. There is an important warning in this statement against prolonged rest cure with confinement to bed for tabetic patients.

There are certain cases of disseminated sclerosis which seem to result from trauma. Mendel reports four which he ascribes to this cause. It is hard to understand how trauma, which causes concussion of the entire central nervous system, can affect the posterior roots or posterior columns alone, as it must do to produce tabes. In a certain number of cases of multiple sclerosis, disturbance of the vascular system seems to have been the commencement of the disease, and it is not difficult to believe that trauma may cause changes in the blood pressure and other disturbances of circulation. The more imperfect vascular supply of the white matter may account for the greater frequency of the sclerotic patches in this part, as the conditions for recovery after a lesion are less favorable. There must, however, be a predisposition, as in many cases disseminated sclerosis does not develop after trauma. This predisposition probably consists of a less capability of resistance of the tissues to hyperæmia, and this predisposition may be congenital or acquired, as from some infectious disease.

SPILLER.

CASES OF TRIGEMINAL SPASM: RESECTION— PROBABLE PRESENCE OF SENSORY FIBRES IN THE SEVENTH NERVE.

By JOHN K. MITCHELL, M. D.,
Philadelphia.

It will not be necessary to state elaborately the history of the patients, a portion of whose symptoms furnish the subject of this brief paper. It is the condition of sensation following the operations for relief, which is interesting.

Briefly, both patients were of the laboring class, both Irish-born, both about sixty years of age. The first, a male, H. M., had a typical example of tic douloureux affecting the supra-orbital distribution on the right side. The usual history of persecution by tooth-drawing was given. Medical measures, especially galvanization, relieved him temporarily when he first applied at the Infirmary for Nervous Diseases. The tic returned in a few weeks as severe as ever, and operation was recommended, and portions of the supraorbital and supratrochlear nerves were removed by Dr. W. W. Keen, November 11th, 1897. Some slight difficulty was experienced in recognizing the former nerve, on account of the hæmorrhage, and to make sure the supposed trunk was carefully followed into the orbit and resected somewhat further back than usual. The portion removed was by inadvertence put in alcohol, which rendered it impossible to make any satisfactory study of its condition, but there was no question of its identity. The patient had at once complete relief from pain, except for two or three slight paroxysms during the fortnight after. The dressings were removed on the third day after operation, and a hasty examination of the condition made. Some degree of anæsthesia was

present for touch and pain in the area usually supplied by the ophthalmic division of the superior maxillary branch. It was noticed then that the loss of sensation was less complete than might have been expected, and seemed to be absolute only in an oval area at the outer canthus of the eyelids and on the upper lid. Later, on the sixth day, opportunity was had for a more careful study. An oval space of about the area of a silver dollar on the temple, at the outer canthus, remained obtuse to touch, but pressure or decided pain was perceivable in this region. Complete anæsthesia of the upper lid persisted. The patient was examined again before his discharge from the hospital, nearly three weeks after operation, and no material change from the described condition was found, except that the area of anæsthesia was a trifle more sensitive.

The second patient, A. K., a married woman, was admitted from Dr. Weir Mitchell's out-service, suffering with pain and spasm throughout the supraorbital distribution, and to a less degree in the superior maxillary division, especially in the palpebral and nasal branches.

Dr. Keen operated on this patient, March 3d, 1898, resecting about half an inch of the supraorbital nerve, drawing the infraorbital out of its canal, and cutting it off also. The former divided rather further from the surface than usual, and at the notch presented several small trunks. The face swelled on the right side after the operation, but otherwise the patient did well.

The excised portions were teased and placed in osmic acid at once, and the interesting changes found will be commented on later.

On investigating Mrs. K.'s face four days after the operation, which was done minutely, on account of the findings in the previous case, we were astonished to discover an even less degree of loss of touch, pain and heat sense than was present in M.'s examination. Indeed, it could scarcely be said that there was more than slight delay or impairment of perception anywhere in the supra-

orbital, nasal, palpebral or labial branches. In the line of the incision made to reach the infraorbital foramen there was poor touch perception, but by the ninth day after operation, when Mrs. K. was discharged, there was neither slowness nor impairment of sensation for any form of stimulation. The touch of the finest filament of thread was instantly felt, and correctly located, everywhere on the cheek, temple, nose, eyelids, and upper lip. On April 20th, the examinations were carefully repeated on both patients—five months after M.'s and seven weeks after Mrs. K.'s operation. In the former, an area of about $1\frac{1}{2}$ by 1 inch on the top of the skull, a little anterior to the vertex and slightly to the right of the middle line, was found to be slightly obtuse to pain. The patient voluntarily stated that the upper part of the right face felt slightly numb. No impairment of sensation could be discovered, even in the line of the cicatrix. Of Mrs. K. the same statement could be made; her perception of touch, pain and heat was rapid and perfect. There was absolutely no lessening of sensibility in any form in either person.

Before discussing possible explanations of these curious results, it may be well to say that, as will be seen, there can be no doubt that the nerves were removed, and the perfection of sensation cannot be attributed to incomplete removal or mistake in operation. Perfect relief followed the surgical intervention, and, moreover, the nerve tissues were microscopically identified. But, even if it were possible to question the entire destruction of the involved nerves, it would still be curious that the restoration of sensation should be so complete, and occur so soon. It is impossible to contend that regeneration of the cut nerves had taken place, for not only was the time too short, but the operations consisted not merely in section, but in the removal of considerable lengths of the nerve trunks.

It is certain that in most cases of neurectomy for tic douloureux anæsthesia in the distribution of the resected nerve follows, but the present are not the only instances

in which an exception to this rule has been noted by the present writer. In a case¹ reported with Dr. W. W. Keen, in which the Gasserian ganglion was removed as the last of fourteen operations, or attempts at operation, for persistent tic, very curious conditions of sensation were present. Before the final operation sensation in the face seemed in an anomalous state, but, as the patient had had the infraorbital nerve cut or removed at three different times, the third division of the fifth resected, the upper jaw bodily removed, and the inferior dental canal laid open and the nerve removed, the face was a tangle of scars, which complicated the examination of sense perception very greatly. The unfortunate man was suffering three or four paroxysms of pain daily. The whole upper right face was extremely sensitive; to touch the eye, to twitch the eyelid, to open the mouth widely, would bring on a furious attack of pain in the *lower jaw*. He was taking morphia steadily, so that various circumstances united to make it difficult to judge exactly the distribution or alteration of sensation; but, so far as could be told, the only part of the face totally anæsthetic to touch was on the right cheek, extending from near the middle line on the right of the nose down to, and including, the ala nasi and out upon the cheek about one and one-half inches, vaguely covering the territory of the infraorbital nerve.

The rest of the face had diminished sensation to touch, but thermic sensation was perfect. After the removal of the ganglion the patient's mental condition was such for nine or ten weeks that no study of the sensation could be made, but, when it became possible, there was no absolute anæsthesia to be found, except between the margins of the wound. Touch-sense was everywhere preserved in some degree, pain-sense was but slightly less than before the operation. He had no spontaneous pain in any part after the operation.

¹ Removal of the Gasserian ganglion for tic douloureux: *Med. and Surg. Reporter*, vol. lxx., No. 12, p. 414.

In the regions supplied by the supraorbital nerve sensibility to touch was diminished as far as the vertex upward, and forward to the median line of the face from the lobe of the ear and the lower border of the inferior maxilla.

The mucous membrane of the lips and cheek on the right side, and the right side of the tongue were also partially anæsthetic. On the right side the sense of taste was entirely lost. The ocular and palpebral conjunctivæ were insensible to touch, but in the right infraorbital region immediately about the scar of the operation upon the infraorbital nerve, there was a small area which was hyperæsthetic.

When seen more than two months after the operation, it was found that an area as large as a half-dollar, with its centre upon the outer third of the right eyebrow, covered a space which was hyperæsthetic to touch, though not to pain, and this although he was unable to move voluntarily the muscles above the brow on this side. Had this patient continued to have *pain* after the ganglion operation, it would have been less astonishing than to find sensation to peripheral stimuli remaining. If the ganglion itself be diseased, whether as a cause or as a consequence of the peripheral nerve trouble, the removal of the ganglion will not affect possible degenerative changes reaching beyond it, and affecting the trigeminal sensory tract in the thalamus, or in its cortical terminus. Disturbances really due to these deep brain changes might be referred to the surface still, in a way aptly comparable to the reference of pain to the extremities of amputated limbs. But, for peripheral touch to be correctly perceived and localized after destruction of the ganglion, or after total ablation of the nerves leading to it from the stimulated part, makes some other explanation than this necessary.

Before discussing possible explanations, something must be said as to the nerve supply of the parts involved. The generally accepted cutaneous distribution of the sen-

sory portion of the fifth gives to the supraorbital branch the supply of the forehead, anterior part of the temple and crown of the head, back to the region supplied by the occipital branches of the second cervical. The eyelids, anterior cheek, nose and upper lip are furnished from the second division, the temples and the rest of the face proper, except the under part of the chin and posterior edges of the lower jaw, being supplied by the third division.

It may at once be said that maps of nerve distribution founded largely upon the somewhat coarse methods of the dissection-table are unreliable. Variations are so frequent they can scarcely be called anomalies. Undescribed anastomoses are often found in operating; whole areas are observed to be furnished with sensory supplies in a manner different from the text-book descriptions. The present writer has before commented upon the frequency, one might almost say the regularity, of unusual distribution in the nerves of the hand and forearm, and the surgical and diagnostic difficulties arising therefrom. But, admitting every probability of error due to such differences, no theory founded on this will suffice to account for such preservation of tactile sensibility as was observed in these cases. One anomaly was very likely present in the first case. The area described as anæsthetic upon the first examination, namely, a region of oval shape at the outer canthus, and covering a small part of the malar prominence, the superciliary ridge and the temple, is usually supplied by the lachrymal branch of the ophthalmic division. The lachrymal leaves the main trunk in the cavernous sinus, and should, therefore, not have been reached by the operation, unless by some reflex effect upon its conducting power caused by the "shock" to the ophthalmic trunk. It is curious that the same, or nearly the same, area was found hyperæsthetic in the third patient after the ganglion removal.

It may be suggested that the nerves cross the middle

line in their final sensory distribution, and that thus each nerve supplies both sides of the face. This is mere supposition; there is no positive evidence of it. If this were at all a constant fact, then, after resection, some loss of sensation should be occasionally observed in corresponding territory on the opposite side of the face; but neither in the present cases, nor in any others, has this been observed, so far as is known to the writer. If both sides are supplied by each nerve so thoroughly as such perfect sense-perception as these cases have would imply, why should there be any anæsthesia after operation at all? Then, too, in these instances at least, the survival of sensation is too complete. In the first there was no impairment a few days after operation; in the second, very little.

What secondary or subsidiary sensory supply could reach these parts? must, then, be the question. The only nerve, beside the fifth, in any way supplying the surfaces of the anterior portion of the head, other than the parts usually deriving their nerve supplies from the upper cervical nerves, is the facial. The facial has been suspected, so to speak, of containing sensory fibres. It is not necessary to refer to the loss of taste occasionally found in cases of facial paralysis, as tending to show the presence of sensory fibres, because this may be directly accounted for by recalling the close anatomical relations of the facial and the chorda tympani, which might well make them both subject to a concomitant lesion, not to mention the near neighborhood of the nuclei of the glossopharyngeal and the nerve of Wrisberg. There are other evidences than these, both pathologic and clinical. To quote some of them: Turner² says: "Certain minor connections are said to exist between the facial nucleus and the corpus trapezoides and the sensory trigeminal root."

Ramon y Cajal³ believes "that the facial nucleus re-

² Edinburgh Hosp. Rep., vol. iv., 1896.

³ Quoted from Mills' "The Nervous System and its Diseases," p. 901.

ceives axis cylinders from the cells of the substantia gelatinosa, which substance accompanies the descending spinal root of the fifth, connection thus being made between the fifth and seventh nerves."

The facial nucleus, in all probability, will be proved to have connections with the thalamus; at present, although this has not been directly proven anatomically, both clinical and physiological facts point that way. Lesions of the thalamus and of the posterior part of the internal capsule have been found, both by experimental and clinical observation, to result in anæsthesia of one side of the body and face; thus here also the fifth and seventh may possibly be in close relation.

As a small piece of negative evidence, might be added the fact that the course of the central neurons of the upper portion of the seventh has not been definitely traced, and may be said to be as yet unknown. Frankl-Hochwart examined, in Nothnagel's clinic, some twenty cases of paralysis of the facial; in eight of these he found sensory or vasomotor disturbances. Sensation was very little altered in any of them, and the disturbance appears, from the brief reports of his observations, to have consisted merely in the slighter grades of anæsthesia. He believes that even these slight changes give ground for the assumption that sensory fibres exist in the facial nerve in man, as it is well known they do in animals.

The cases here reported strengthen, from clinical observation, the probability of this view. No doubt other observations will be made confirming these. What would be of most importance would be to discover peripheral sensory changes of decided or constant character in cases of facial paralysis. Could a series sufficiently large of cases of total paralysis of the seventh be observed without any of them presenting any sensory changes, such a mass of negative evidence might be held to outweigh the small positive results; but till then the probable explanation of such conditions as these here reported may be held to lie

in the presence of sensory fibres in the seventh nerve.

For the sake of the interest, although somewhat beside the chief subject of this paper, Dr. William G. Spiller's comments on the pathological findings in the nerves removed from the second case are added, by his courtesy:

Many, possibly most, of the nerve fibres of the infra-orbital nerve, when separated from one another by teasing and stained by a one per cent. solution of osmic acid, are found to contain numerous black balls, approximately of the same size. These are nearly equidistant from one another, and are located along the edges of the fibres, leaving, as a rule, the centres free from such accumulations. When the focus of the lens is changed, so as to bring other portions of the fibres into view, black balls are apparently found within the centres, but these are probably along the superficial and deep portions of the fibres. The medullary sheaths are thus broken into numerous masses of nearly equal size, occupying the normal position of the myelin sheaths. It is exceptional to find masses of degenerated myelin of a size so large as is frequently seen in degenerating fibres. Similar lesions are found in the supraorbital branch of the fifth nerve. Inasmuch as these nerves were taken from the living subject, and placed immediately afterward in osmic acid, these myelin balls cannot be regarded as artifacts due to surgical interference. Such interference causes a breaking of the fibres into irregular masses, but probably not the fragmentation of the myelin into numerous balls.

Sections cut with the microtome and stained with carmine and Delafield's hæmatoxylin show more or less round cell infiltration about the small vessels. The coats of the smallest vessels are not notably thickened, but one vessel of larger size, found in some of the sections, presents a thick media and a somewhat proliferated intima. In some of the nerve fibres pale purple bodies are found, which resemble the amyloid bodies, and lend much support to the view that the latter are degenerated nerve

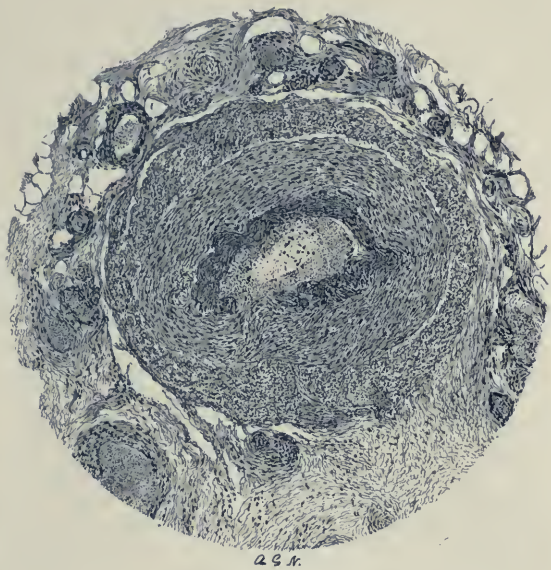


FIG. I.

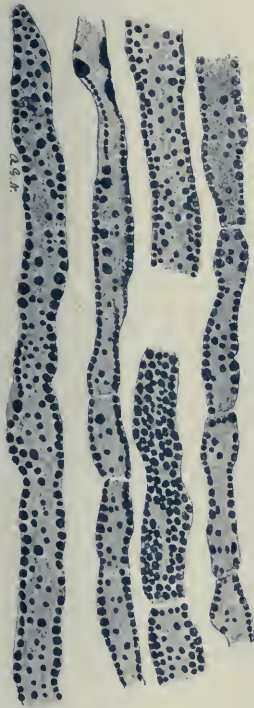


FIG. III.

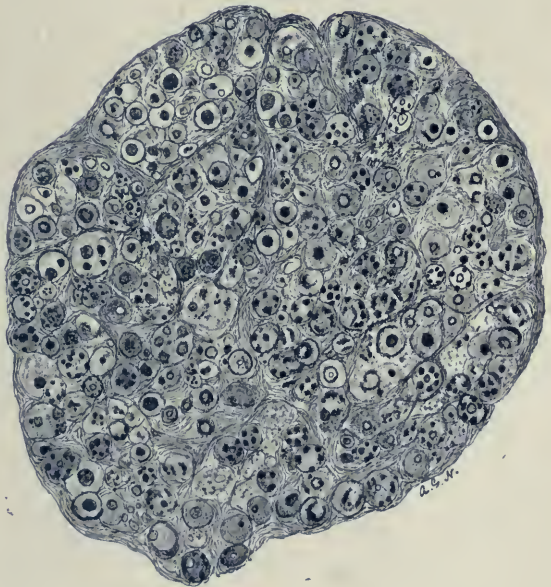


FIG. II.

FIG. I. Thickened vessel from the infraorbital nerve.
 FIG. II. Infraorbital nerve stained by the method of Weigert. Balls of myelin are seen as chains of beads about the axis cylinders.
 FIG. III. The medullary sheaths are broken into numerous balls.

fibres. Most of the nerve fibres contain an axis cylinder, though in some this cannot be seen.

The hæmatoxylin method of Weigert, used on transverse sections, reveals the presence of the myelin balls within the nerve fibres in the same manner as the osmic acid shows them in longitudinal sections. They appear as a circle of beads about the axis cylinder. The nerve fibres in some bundles appear to be fewer than normal, though this may be due to degeneration of the myelin, and, therefore, imperfect adaptability to the hæmatoxylin stain.

133. ENCEPHALITIS AND LATE EPILEPSY Jas. G. Kiernan (Alienist and Neurologist, 18, 1897, p. 168).

Epilepsy occurring after 25 and that due to encephalitis have points of special interest in common. Epilepsy following on the various forms of infantile encephalitis is more apt to be accompanied by atrophic phenomena. It reacts badly to the bromides; mental symptoms replace under the bromides the convulsions. The tendency to impairment of the circulatory innervation of the extremities is increased. The bromic dermatoses appear with great frequency. A nocturnal mental type, resembling somnambulism, takes the place often of the convulsion. Encephalitic epilepsy, while in many cases possibly Jacksonian at the outset, but too often becomes an epileptic constitution, with all the phenomena of idiopathic epilepsy. After the age of 25, and most frequently between 35 and 40, in persons with no decided neurotic heredity, and in most of whom lues can be excluded, occurs an epilepsy which resembles that from encephalitis. As a rule, in these cases there has been a precedent period of nervous exhaustion, attended by vertiginous states as its later development. These states are often preceded or followed by anomalous sensory disturbances, compared by the patient to "waves." There is loss of or dazed consciousness, with or without motor explosions. Some of these states, even when with consciousness, are attended by localized jerkings of groups of muscles. All these phenomena are clearly due to toxins resultant on nerve exhaustions. Normally, the toxins produced in the body are eliminated by various channels. When any of these emunctories are interfered with the phenomena of auto-intoxication appear. The alterations are peculiarly suited for treatment of states due to toxins. By destruction of the toxin through stimulus of hepatic action and elimination they prevent its accumulation and the resultant phenomena. The alkaline bromides do not seem to exert this influence. Of late metallic bromides have been united in two compounds—the liquor (arsen. auro) and liquor arsen. auro and hydrarg. These, alternated weekly, give in the types mentioned undeniably excellent results.

This has no contraindication from the bulk of dose, is readily given with meals, and in the quantities needed is exceedingly economical. It tends to diminish retrograde metamorphosis in the intestine, and hence destroy the undue accumulation of gas. FREEMAN.

HYPERTROPHIC NODULAR GLIOSIS.

By JOSEPH SAILER, M.D.,

Associate in the Pepper Clinical Laboratory; Pathologist to the Pennsylvania Training School for Feeble-Minded Children, to St. Joseph's and to St. Christopher's Hospitals.

Hypertrophic nodular gliosis of the brain does not appear to have escaped entirely the notice of the older writers, although their descriptions of, or, rather, their allusions to, this condition are so vague and unsatisfactory that it is impossible to be altogether sure of this fact. Bourneville was the first to give a clear description of the disease, and his earliest case was published as recently as 1880. A number of cases have since been published, but only a few have been carefully studied, and it appeared desirable, therefore, to place the following example upon record:

J. H., white, male, was admitted to the Pennsylvania Training School for Feeble-Minded Children, on November 2d, 1888. An imperfect history was obtained from a member of the Board of the Children's Aid Society. The boy's father was born in Massachusetts; he was a plumber, and addicted to the use of alcohol. His mother was born in Ireland, and seems to have had no neuro-pathic condition; it is stated that she died of cancer of the breast at the age of 50. Nothing is known of the grandparents. The patient was the last of four children; two brothers are dead; cause, unknown; one sister was placed in a hospital for the insane at the age of thirteen. The patient was born at full term after a normal labor. He

NOTE.—I desire to express my thanks to Dr. Martin W. Barr and to Dr. Frank White of the Pennsylvania Training School for Feeble-Minded Children, who have kindly placed the clinical history of this case at my disposal. The microscopical work has all been done at the Pepper Clinical Laboratory.

was apparently healthy until ten months old, when he began to have occasional spasms, which occurred more frequently as he grew older. He did not walk until four years of age. His gait was always rather slow and unsteady, and the power of coördination poor. He was able to say two or three words, but those were spoken very indistinctly.

The following notes were made on September 10th, 1896:

"J. H., aged fifteen, low-grade idiot and epileptic, body fairly well nourished, muscular system poorly developed, cutaneous reflexes apparently normal, cremasteric reflexes increased, patellar reflexes exaggerated, tactile sensibility apparently normal, thermal sensibility uncertain. The eyes are myopic; the pupils react to light. Hearing is probably good. The head sphaicephalic but symmetrical; Romberg's symptom is present. Since admission he has been treated for epilepsy, purulent otorrhœa and prolapse of rectum." The following notes are given in regard to his general condition: "He is totally dependent in every way, being unable to feed himself. When food is placed in his mouth he makes little effort at mastication, so that it is necessary to give him liquid or soft diet. He sometimes drinks a large amount of water at one time, but it seems to run into the stomach from gravity, there being no effort on his part to swallow, and it does not seem to alleviate his thirst. He was subject to merycism. There was often considerable protrusion of the rectum. He was an onanist; the habit being apparently more pronounced before an epileptic attack. For some hours previous to the outbreak of a spasm he would bark like a dog. When taken with a fit, if standing, he would fall instantly, the right side of the head striking first. During the seizure the muscles of the right side of the face showed quick, spasmodic movements; the left side remained in tonic contraction; the right hand was open and the left clenched, and the muscles of the left forearm continued spastic for some time following the spasm. After the spasm he would laugh in a silly fashion

an hour or more. Before the spasm he was irritable and restless, but apparently more intelligent than at other times. His last illness began in December. It commenced with an almost constant succession of spasms, 63 occurring in two hours. They were general, but, as a rule, more severe on the right side; the head was turned to the right; the left arm was rigid and lay in the bed; the right arm was rigid and elevated; the left leg twitched, the right was rigid; the hands were flexed at the wrist; the fingers fully extended. The spasms decreased to about 10 or 12 per hour, and in the course of 24 hours the patient died from exhaustion."

The *autopsy* was performed on the 10th of December, 1896, about 24 hours after death. The following notes were taken:

Body of boy moderately emaciated, no rigor mortis, no postmortem lividity. The testicles were still in the inguinal canal; there was a bruise on the forehead and a deep cut on the chin, penetrating almost to the bone. There were numerous petechiæ on the surface of the abdomen. The meninges of the spinal cord contained a considerable amount of fat; they were not injected, and there was no abnormal collection of cerebrospinal fluid. The substance of the cord was firm. The skull was slightly thickened; the dura mater was firmly adherent, but there were no signs of inflammation. The pia mater was smooth, transparent, and stripped easily. In the cortex of the cerebral hemispheres were a number of sharply circumscribed areas much denser than the normal brain tissue. These were pale, slightly protuberant, and the large ones slightly depressed in the centre. When incised, the surface of the section was seen to be grayish white, firm and dry. The nodules appeared to extend a short distance into the white matter, but the distinction between the medulla and the cortex was preserved. The ventricles were not dilated, but upon the floor of the lateral ventricles were a number of small, white nodules, a few mm.

in diameter, projecting distinctly above the surface. The circle of Willis was normal. There was a hemorrhage into the floor of the fourth ventricle. The whole brain appeared to be larger than normal. The abdominal organs were normal in arrangement. There were some pleural adhesions and moderate hypostatic congestion in both lungs. The heart was small; the left ventricle moderately hypertrophied, and there was slight sclerosis at the bases of the aortic valves; the mitral valve admitted one finger; the aorta was elastic and smooth. The heart contained numerous chicken fat clots. The surface of the duodenum was covered by numerous small nodules of varying size, quite firm in consistency. The rest of the intestinal tract showed no gross morbid changes. The mesenteric glands were greatly enlarged. Both adrenal capsules showed advanced fatty degeneration of the cortex. The right kidney contained a huge tumor-like mass about 7 cm. in diameter, that seemed to replace a portion of its substance, and numerous small nodules. The surface of the tumor was smooth and white, and did not exude any juice; the growth was sharply circumscribed; its consistency was exceedingly dense. The small nodules were similar in character. The capsule of the kidney was adherent; the cortex was pale, narrowed, granular, and contained numerous cysts, and the renal vessels were injected. The left kidney contained small nodules, in all respects similar to those of the right, and the kidney substance showed the same changes. The liver was greatly enlarged. The left lobe extended to the left posterior axillary line, overlying the spleen; upon the surface there were numerous whitish discolorations. Upon section, it appeared to be normal. The mucous membrane of the stomach was thickened, rugous and somewhat pale. The mucous membrane of the duodenum contained a number of small, hard nodules. The rest of the intestinal tract was normal. The pancreas was soft, and showed no changes. The bladder contained turbid urine, probably

due to precipitation of the urates, for the mucous membrane was smooth. The thyroid gland was apparently normal. The thymus gland was present. The thoracic and cervical lymph glands were enlarged. The tumors of the kidney were composed of spindle cells, some with round or oval nuclei, and others with rod-shaped nuclei that resembled non-striated muscular fibres.

Microscopically, it was seen that the growths infiltrated the surrounding tissues, and a number of the renal tubules in the neighborhood exhibited proliferation of the epithelial cells. The tumors were therefore diagnosed adenosarcomata of the kidney, although neither cartilage nor striated muscular tissue was found. According to Birch-Hirschfeld, these tumors commence in fetal life, and may attain considerable size in childhood, and ultimately, if death does not occur too early, give rise to metastasis. Sections through the wall of the duodenum showed that the various layers were normal, with the exception of the mucosa. In this the tumors were represented by masses staining a diffuse blue with hæmatoxylin; the central part was pale; the peripheral darker. There was no distinct structure to be found in these masses, but, as they faded into the surrounding tissue, small, round cells became more distinct. The condition represents hyperplasia of the lymph follicles, possibly sarcomatous in nature, which accounts for the distinct involvement of the capsule, although the extreme degeneration of the tissue renders it impossible to speak positively. There was no evidence of metastasis in any other part of the body. A more minute examination of the brain after hardening in Müller's fluid showed the following changes: The pia stripped readily; the nodules were slightly elevated, hard, paler than the surrounding tissue, and the surface exhibited slight granulation. The membranes were not thickened nor congested. When incised, the dense tissue involved all of the cortex, which was usually increased in thickness, and extended a few millimeters into the white substance.

The distinction between cortex and medulla was maintained, chiefly by the greater retractibility of the latter. There was no appearance of congestion in the morbid tissue. The areas of gliosis are very irregular in shape and very unequal in size, ranging from 0.5 cm. to 7 cm. in diameter. They are distributed as follows:

Left Cerebral Hemisphere.—Convex surface in the superior frontal convolution; at the anterior end a small nodule; in the middle a group of three small nodules; at the posterior end a large nodule that extends to the median surface. In the second frontal convolution, at the anterior end, a large sclerotic patch involving four small convolutions; just back of these is a smaller mass, and at the posterior end a large mass that extends along a small annectant gyrus, and involves the ascending frontal gyrus. In the third frontal convolution the operculum is normal; the anterior part is occupied by a huge sclerotic mass, 5x7 cm. in extent, involving a number of secondary gyri. In the ascending frontal convolution there is only the nodule that extends from the second frontal. In the ascending parietal there is a small nodule about the centre, near the fissure of Rolando, and a larger one near the longitudinal fissure, and a still larger one in the posterior portion of the superior parietal, about 3x6 cm. in area. The supra-marginal gyrus contains a small mass in the anterior limb, and the whole of the angular gyrus appears to be converted into a single sclerotic mass, perhaps the largest in the brain. The occipital lobe is composed of a number of nodules of various sizes, separated by small areas of softer tissue, none of the convolutions being entirely normal. In the first temporo-sphenoidal convolution there is a small nodule in the posterior end; the second is completely sclerosed anteriorly; in the third there is a small nodular mass at the anterior, and two at the posterior extremity; in the fourth there is a mass of gliosis, posteriorly extending into the occipital lobe. The fifth convolution is apparently normal.

The Internal Surface.—There are large nodules in the anterior and posterior part of the first frontal convolution, numerous nodules in the quadrate lobule; a large mass in the posterior portion of the cuneus, and a few nodules on the orbital surface of the frontal lobe. The cornu ammonis is apparently normal.

Right Cerebral Hemisphere.—In the superior frontal convolution there are several nodules in the anterior portion, a large mass in the middle, and another posteriorly. In the third frontal convolution there is a mass in the anterior portion. The operculum is not involved. There is a nodule in the lower extremity of the ascending frontal convolution. In the superior parietal there are a few nodules posteriorly. The supramarginal gyrus is involved throughout. The occipital lobe resembles that of the left side. The anterior portions of the first, second and fourth temporo-sphenoidal convolutions contain some nodules. The cornu ammonis is normal. On the internal surface there is a mass of gliosis in the posterior part of the cuneus. The calloso-marginal convolution contains a few scattered nodules. There is a large mass in the posterior part and another in the middle of the superior frontal convolution, and a few small nodules in the orbital extension of the second frontal. The cerebellum, pons and medulla are apparently uninvolved. In the interior of the brain, areas of sclerosis large enough to be detected with the naked eye cannot be found, excepting the nodules already described upon the surfaces of the ventricles. The third and fourth ventricles are free, and the basal ganglia, as far as examined by horizontal section through the brain, are not affected. The pyramids and olives in the medulla are quite distinct. The peduncles and corpora mammillaria are of equal size. The cerebellum is of normal size and consistency. There are no gross lesions of the cord.

The microscopical appearances were as follows: When the sclerotic areas were stained with hæmatoxylin and eosin the following changes were observed: The neuroglia

was composed of coarse fibres, forming a coarse-meshed reticulum, or else arranged in bands, or twisted into a sort of spiral form; these changes were particularly noticeable either just beneath the pia mater, or about the junction of the gray and white matter; the neuroglia cells were somewhat smaller than normal, irregular in outline, and moderately increased in number. In those situations where the sclerosis was greater, the vessels were greatly increased in number; beneath the pia, however, the sclerotic areas seemed to be less vascular than normal. Nearly all these blood vessels were distended with blood, a result, I suppose, of the violent muscular effort occurring during the status epilepticus. They appeared to be much larger than normal, and the walls often showed proliferation of the nuclei. The perivascular spaces were frequently distended, sometimes exceeding in width the diameter of the vessels. Occasionally they were filled with a delicate reticulum, although it could not be determined that this was composed of neuroglia fibres. There was no accumulation of round cells in the perivascular spaces, but occasionally large cells could be seen that contained granules staining black with osmic acid. These are probably connective tissue cells that have absorbed fatty detritus, perhaps resulting from a degeneration of the myelin sheaths. When the vessels were cut longitudinally, it could be seen that they were moderately tortuous, but they did not show any aneurismal dilations. In sections stained by Rosin's method they appeared quite red, partly due to the staining of the red blood vessels; but in the more sclerotic areas the walls also appeared to have taken this color, and it may be that they have undergone hyalin degeneration, particularly as the sections stained by Van Gieson's method showed the same peculiarities. Amylaceous bodies were not observed in the cortical lesions. The nerve cells stained fairly well; they were present in all the areas of sclerosis, excepting those few where the bundles of neuroglia were arranged in wavy bands or spirals and per-

meated by large and numerous vessels. Neither by carmine nor hæmatoxylin did they show any signs of degeneration, but they appeared to be more irregularly placed than usual. This alteration was still more pronounced in the sections that had been stained by Nissl's method, in which it could be seen that the apices of the pyramidal cells pointed in all directions. They were very numerous, even more so, apparently, than normal, and stained rather deeply; their processes were slightly tortuous, but there were no definite nuclear changes and no alterations in the protoplasm. In position they seemed to be abnormal, large cells being found in the second layer as well as the third. A number of sections were also stained by Berkeley's modification of Golgi's method. The neuroglia cells appeared to be very numerous, and impregnated very distinctly. The pyramidal cells were only found in a few sections, in which they seemed to be perfectly normal. The method is, however, so unsatisfactory for pathological purposes, that its only value in the present instance is to show that normal cells exist in the sclerotic areas. In some parts of the cortex certain peculiar bodies were observed in considerable number, that were circular or pear-shaped and very pale, appearing like vacuoles in the midst of the neuroglia tissue. Each contained a small, faintly staining, round nucleus in the centre, and probably represented a degenerated cell; but whether they are derived from the ganglion or the neuroglia cells, it is quite impossible to say. A number of sections were stained by the Weigert or Pal methods. In nearly all these the tangential fibres of the cortex were absent. Toward the white matter the cortex remained perfectly pale, with the exception of a few delicate fibres that passed irregularly through it. When the gliosis invaded the white matter, it did not in general cause complete destruction of the myelin sheaths, but the distance between the individual fibres was considerable, and gave rise

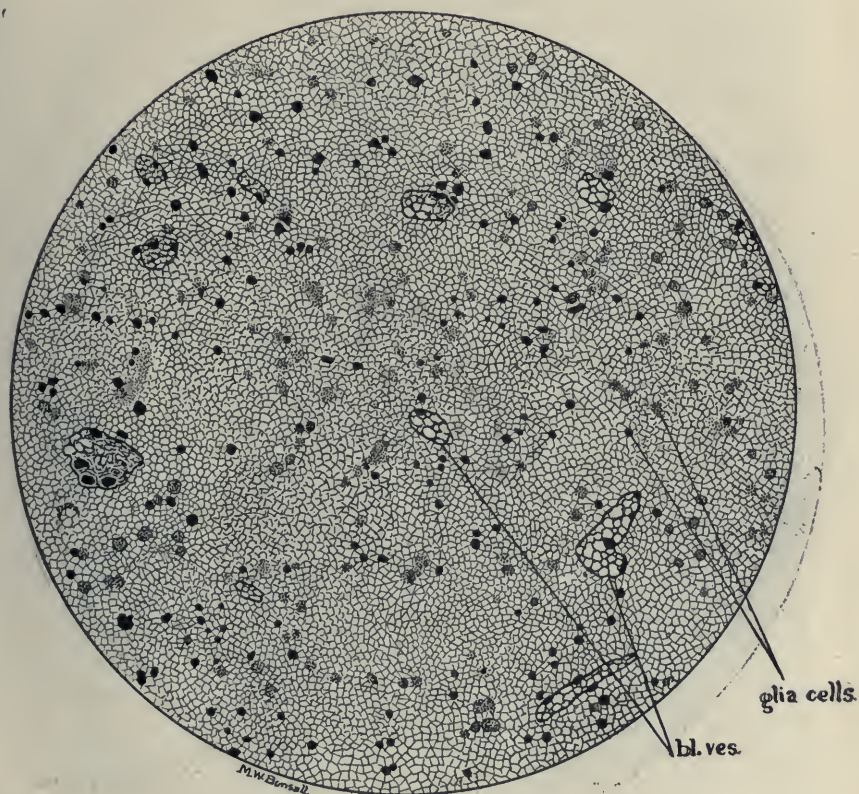


FIG. 1

Section through a sclerotic area in the third frontal convolution of the right side. The focus is about 4 mm. in diameter and situated at the junction of the cortex and white matter. It contains an excessive number of blood vessels, the neuroglia cells are more thickly placed than is normal, and the neuroglia fibres are coarser and form a looser meshwork. (Hæmatoxylin and eosin. Zeiss DD; oc. 3.)



Fig. 2

Section through a nodule in the floor of the right lateral ventricle containing hyaloid bodies. Van Gieson's stain. (Zeiss AA; oc. 3.)

to an extremely feathery appearance at the junction of the white matter and the cortex. In these areas the sections appeared to be much paler than in other parts. The fibres of association could not be made out, and were probably largely lacking. Where the sclerosis was most advanced no myelinated fibres could be found. No degenerated fibres were found by Marchi's method, but a few cells containing black granules were observed in the perivascular spaces.

The nodules upon the floor of the lateral ventricles consisted of almost pure neuroglia tissue, arranged in bands, or in whorls twisted about the centre of the nodule. The blood vessels were greatly increased in number throughout the majority of these sclerotic areas, and in the sections stained by Van Gieson's method, exhibited the same changes as those in the cortex. In one of the nodules on the left side, there were a number of peculiar, irregular bodies, that were usually circular in shape, although sometimes slightly elongated, and showed a distinctly concentric structure. They stained deep blue with hæmatoxylin, remained a deep brown color in the sections stained by Weigert's method; they retained the stain by Gram's method, and with thionin and acid fuchsin stained bluish green with a delicate red border; they stained a deep red by Van Geison's method, and bright red with gentian violet. They were not soluble in alkaline or acid solutions. I am inclined to believe, on account of their situation beneath the ependyma and their peculiar staining reactions, that they represent agglomerations of altered amyloid material. They do not, however, stain brown with iodine. I have observed exactly similar bodies in an area of softening in the brain of a child that died of tubercular meningitis, and others somewhat similar in a case of paralysis agitans with marked arterial sclerosis. They appear, therefore, to indicate degeneration of nervous tissue, a view held by Stroebe and also by Dagonet, who regards them as a derivative of cerebrin. Beyond

these nodules the hyperplastic neuroglia tissue extends deeply into the optic thalamus, showing the same thickening of the fibres and coarse meshing that was found in the sclerotic areas of the cortex. The ependyma covering the nodules was considerably wrinkled, giving, in cross section, a slightly papilomatous appearance; it was composed of a single layer of polygonal cells with deeply staining nuclei, that showed no evidences of proliferation, such as are present in neoplastic and inflammatory processes; the appearance bore a striking resemblance to that often seen in the intima of sclerotic arteries, and was probably due to retraction of the neuroglia tissue. In the cerebellum quantitative changes were distinctly seen. The cells of Purkinje were sometimes widely scattered, sometimes close together. They stained bright red with eosin; the nucleus showed a faint bluish tinge, was irregular in outline, and apparently contained very little chromatin. The medullated fibres were much fewer than ordinarily observed, and none at all were found in the cortical layer. The vessels in the cerebellum were not particularly numerous, and did not show the perivascular spaces. The pons was apparently normal. Along the edge of the left pyramidal tract in the medulla was a small area of sclerosis, sharply defined from the rest of the tract. The neuroglia cells appeared to be increased in number, and there was the same reticular arrangement of the neuroglia fibres. Otherwise the process resembled more closely a patch of insular sclerosis. In the spinal cord (lower cervical region) no distinct changes were noted, excepting on the left side, just outside the anterior cornua, where there were a number of dilated blood vessels, somewhat tortuous in their course, and a slight proliferation of the neuroglia tissue, staining red with acid fuchsin. Sections stained by the Weigert, Pal and Marchi methods, and by carmine, failed to show any trace of degeneration. The ganglion cells of the anterior cornua were normal. The central canal still existed as a microscopical channel.

An examination of the literature for similar cases has yielded the following results. It must not be understood, however, that all the cases of which I give abstracts were reported as tuberous gliosis. Some have been included under the title of hereditary syphilitic disease, and others have been described as cases of insular sclerosis.

The negative testimony is not uninteresting. Echeverria, whose book upon epilepsy appeared in 1870, and was the best monograph that had hitherto been published upon the subject in America, makes no mention of any lesion similar to this, although he describes quite extensively the pathological change found in epileptic brains:

Case I.—Bourneville. The patient was a female without neuropathic heredity. Convulsions commenced in infancy, and at the age of two years assumed the form of typical epilepsy. She never learned to walk nor talk, and when she was admitted to the hospital, at the age of fifteen years, it was found that the right arm and leg were flexed and partially atrophic. The epileptic attacks were Jacksonian in type, the spasm commencing in the right eye, which was drawn up and to the right; then rigidity of the right arm, followed by clonic spasm of the right arm and eyelids. At the autopsy the brain was large and slightly asymmetrical, the right peduncle and corpus mammillare being larger than the left. The pyramids were united with the olives; the pia mater was delicate and slightly adherent over the focal lesions. In the cortex of both cerebral hemispheres were numerous rounded masses of various sizes, white, opaque, firmer than the brain substance, and slightly prominent, with a shallow umbilication in the centre of the larger ones; the cornua ammonis were normal. The lungs were pneumonic; the left ventricle of the heart hypertrophied, and white nodular masses, apparently cancerous in structure, were found in both kidneys.

Case II.—Bourneville and Brissaud. The patient was

a male; two other children were healthy. The mother had had convulsions during pregnancy. The child learned to walk at the age of two years, but could not speak. He had frequent epileptic attacks, during which he was cyanosed, and later this condition became permanent. There was a loud cardiac murmur. Death occurred at the age of four years, apparently from cardiac insufficiency. There was a large area of softening in the right frontal lobe; over which the pia mater was adherent. The sclerotic areas were found in both hemispheres, but were more numerous on the left side. There was a congenital lesion of the heart. Microscopic examination of the brain substance showed the absence of nervous elements in the sclerotic tissue. The glia cells were increased in number, but were somewhat irregular in shape. There was no sharp line of demarkation between the sclerotic and normal tissue, but the ganglion cells in the neighborhood of the lesions were granular and yellow; the blood vessels were few but large, and Brissaud, who made the examination, declares that medullated fibres were absent in the lesions, but he used only the carmine stain.

Case III.—Bourneville and Bonnaire. The patient was a male, the tenth of thirteen children, of whom only three others, all of whom appeared to be normal, lived for any considerable time after birth. There was neuropathic family history. The child was apparently normal for the first five months, and then received a severe injury to the head, which was followed by a notable change in its disposition. Convulsions, during which the child would become rigid for about 15 minutes, without loss of consciousness, commenced at the age of seven and a half months. He did not walk until two years and a half old, could not talk, recognize his parents nor feed himself. He died at the age of five years. The brain weighed 1,040 grms.; the dura mater was adherent to the calvarium. The pia mater was injected, but not adherent. The sclerotic nodules were found in the cortex and in the

corpus striatum. Numerous round tumors were found in the kidney, which proved to be encephaloid sarcoma. There were no lesions in the isthmus or cerebellum, and the ventricles were not dilated.

Case IV.—Bourneville and Bonnaire. The patient was a male, the fourth of seven children, only three of whom lived beyond the age of infancy. There was marked neuropathic heredity, and both father and mother had had skin eruptions. The child was born at the sixth month, and had signs of scrofula. Convulsions were first noted in the sixth week, and recurred frequently. At the age of two years there was paresis of the neck and arms, but this disappeared two years later. The child never could walk without support, and there was a tendency to fall forward. It was an imbecile and filthy in its habits, the only manifestation of intelligence being the occasional expression of pleasure. Death occurred at the age of five years in status epilepticus. The sclerotic nodules were found in the cortex and basal ganglia. The occipital region of the brain was slightly flattened. The kidneys contained numerous serous cysts and raised tumors, that were soft and white.

Case V.—Bourneville and Bonnaire. The patient was a male, with neuropathic family history; two children were dead, two others lived, and were apparently healthy. There was no history of syphilis or injury. Convulsions commenced at the age of five years and nine months. The sclerotic nodules were found in the cortex of the hemispheres and in the caudate nuclei. The kidneys contained cysts and some large, firm tumors.

Case VI.—Hartdegen. The patient was a male, asphyxiated at birth and unable to nurse or to swallow. The anterior fontanelle was dilated, and there was a lumbosacral spina bifida that became infected, giving rise to a leptomeningitis, apparently the cause of death, which occurred at the age of forty-eight hours. Numerous sclerotic nodules were found on the surface of the hemispheres,

which were firmer, paler and dryer than the brain substance. Smaller nodules were found in the walls of the ventricles. The thoracic and abdominal organs were normal. Microscopically, the sclerotic areas consisted of a finely granular tissue containing delicate fibres. The sclerotic lesions contained numerous ganglion cells that were irregular in shape and abnormal in position, some of the largest being found in the most superficial layer of the cortex. The nodules in the lateral ventricles consisted of large cells with numerous processes, that were mingled with fibres of connective tissue, extending downward from the ependyma. He regards these growths as *glioma ganglionare cerebri congenitum*. The ventricles were dilated. The convolutions were normal in arrangement.

Case VII.—Pollak. The patient was a female, whose mother had had severe headache and attacks of dizziness during pregnancy. Two other children were healthy; one had died of epileptic attacks. Convulsions occurred four days after birth, accompanied by fever and œdema of the scalp; this continued four days, after which the child seemed parietic, no spontaneous movements being observed excepting a slight twitching of the facial muscles when loud noises were made. Deglutition was always difficult, and the patient showed a marked aversion to liquids, especially water, never drinking, although she had constant polyuria. In spite of this, she was well nourished. As she grew older, she manifested pleasure when shown bright objects or when she heard music, and recognized her father's voice. There were occasional attacks of trembling, and paradoxical contraction was observed in the tibialis anticus and quadriceps extensor. There was nystagmus, divergent strabismus and unequal pupils; the fixing eye was in extreme myosis and the other mydriatic. During the second dentition muscular atrophy commenced in the feet and hands. In the fourth year of her age chronic cramps occurred in the limbs; there were gnashing of the teeth and the signs of hydrocephalus, fol-

lowed by death in a few months. At the autopsy it was found that the dura mater was adherent, the pia mater thickened and partially adherent. Pale, hard elevations were observed in the cerebral hemispheres, anterior to the ascending parietal convolution, and over these the pia mater was firmly adherent. The corpus callosum was thickened and nodular. The ventricles were rough, and the basal ganglia and spinal cord contained areas of sclerosis. No microscopical examination was made. Pollak calls the case one of congenital disseminated sclerosis, and claims that he made an ante-mortem diagnosis, although he appears to be unfamiliar with the nature of the process. Two years previously he reported a living case with similar, but less severe, symptoms.

Case VIII.—Bruckner. There was no neuropathic heredity, no history of injury or of syphilis. The patient was a girl, who commenced to speak in her second year and to walk in her fourth. At school she learned to write with great difficulty, and was always shy and timorous. She was never able to calculate. The first epileptic attack occurred in her ninth year. After this fits recurred occasionally, and there was some disturbance of the gait, which later improved. At the age of eighteen years, the patient became maniacal, and then passed rapidly into a state of apathetic idiocy, although sensation, motion and the vegetative organs remained normal. A year later epileptic attacks recommenced, but were quite infrequent. At the age of twenty-two years the patient died of phthisis. At the autopsy the brain was found to be large and to contain numerous circumscribed foci of hardening, the larger ones having depressed centres. They involved principally the gray matter, but also extended into the white substance. Microscopically, a coarse fibrous connective tissue was found, especially just beneath the pia mater, where it assumed the form of a band of wavy fibres. In places ganglion cells were observed, included in a fine-meshed reticulum, that were apparently normal, but more

thickly placed than usual. The blood vessels were characterized by wide perivascular lymph spaces. The ventricles of the brain were distended, and from the ependyma a fine fibrous stroma extended inward, containing holes filled with large, glassy, round cells, between which were some chalky deposits.

Case IX.—Pozzi reports the case of a man admitted to the hospital, at the age of sixty-eight years, suffering from dementia, with occasional outbursts of maniacal violence, alternating with states of melancholic depression. There was a history of epilepsy, but nothing further could be ascertained about the patient. Shortly after admission, and two months before death, the epileptic attacks recurred with great frequency, and death was apparently due to "status epilepticus." At the autopsy the skull was thick, the dura mater partially adherent, the pia mater injected, but not adherent, and there was some subarachnoid effusion. In certain regions the convolutions were small, hard and granular; in others they were enlarged, hard and smooth, the enlargement being nodular in character. Brissaud examined the specimens microscopically, and reported that the hypertrophic areas resembled cirrhosis, the neuroglia forming a dense, non-vascular mass, harder than the brain tissue. The ganglion cells were not degenerated, but rarer than usual. The large motor cells could not be found. There was no disturbance of the normal succession of the layers of the cortex. The atrophic convolutions were similar in character, excepting that the neuroglia seemed to be denser and the ganglion cells even fewer. He also found certain round masses in the midst of the neuroglia, that he regarded as colloid in nature.

Case X.—Simon. The patient was a female, with neuropathic family history. At the age of two and one-half years she was admitted to the hospital, and it was noted that she was microcephalic and showed contractions of all four limbs. There was continual agitation of the

muscles of the face and limbs and frequent attacks of epilepsy. The intelligence was almost nothing. The digestive functions, excepting deglutition, were normal. Death occurred one month after admission. At the autopsy two sclerotic nodules were found in each hemisphere, just in front of the fissure of Rolando. The convolutions in the neighborhood were atrophic. The sclerotic tissue extended two or three millimeters into the substance of the brain, and the microscopical examination showed proliferation of fibrous tissue, the destruction of the cells and of the myelin sheaths. There were secondary alterations in the pyramidal columns of the cord.

Case XI.—Schule. The sex of the patient is not given, and there is no family history. The epileptic attacks commenced at the age of three years. The patient was not a total idiot, and learned something at school. There was right-sided facial atrophy, the gait was normal, Romberg's sign was present, the speech was slow, and the hearing of the left ear imperfect. Death occurred in status epilepticus, at the age of sixteen years. The brain weighed 1,390 grams. The dura was adherent to the skull and the pia mater over the projecting sclerotic mass in the frontal region. The brain otherwise exhibited typical nodular sclerosis.

Case XII.—Buchholz describes the specimen from a case. The brain contained some nodules upon the surface of the convolutions and some cysts in the brain substance. There was round-cell infiltration around the vessels, as if the processes were inflammatory in nature. Some of the nodules were composed of true connective tissue, with spindle-shaped nuclei, and a similar tissue was found in the cysts. It is stated that some of the fibrous bands represented degenerated vessels.

Case XIII.—Thibal reported another case from the service of Bourneville, but I have been unable to obtain the original paper.

Cases XIV., XV., XVI.—Buchholz reports three doubtful cases. Two of the patients were epileptic males; in the brains of both of whom increase of the neuroglia cells and hyperplasia of the neuroglia tissues were found, and also degeneration of the ganglion cells. The changes were principally found in the cortex, but sometimes affected the white substance, and occasionally led to cavity formation, in which case compound granular cells were usually present. One of the brains weighed 1,575 grams. The third patient was a paranoiac woman, who had been married; her brain presented similar changes.

Cases XVII. to XXII.—Wilmarth. Six cases are mentioned as having been found in the brains of epileptics and idiots at the Training School for Feeble-Minded Children. In some, the situation of the sclerotic nodules in the motor region explained the symptoms. The descriptions are very meagre and unsatisfactory.

Case XXIII.—Henoch. The patient was included in a series of doubtful cases of cerebral syphilis. The child was admitted to the hospital at the age of two years, and presented a peculiar alternation of slyness and stupidity. There was spina ventosa, but no disturbance of motility. Death occurred from diphtheria. At the autopsy a number of rough tumors, about the size of cherries, were found in the substance of the brain and cerebellum. A similar growth was also found in the upper portion of the left kidney. There was some periostitis upon the surface of the tibia, and the tumors were declared by a pathologist to be gummata.

Case XXIV.—Berdez. The patient was a male, without neuropathic family history. Other children in the family were perfectly healthy. At the age of four months convulsive attacks occurred, in which the child became slightly rigid, and there were movements of the eyes, followed by periods of loss of consciousness. Later, these became more typically epileptic. The child never talked nor gave any signs of intelligence, and could not walk.

At the age of two years, when admitted to the hospital, the head was found to measure fifty-two centimeters in circumference. The pupils were normal; there were no muscular atrophies nor choreic movements of the extremities. A diagnosis of hydrocephalus was made, and craniotomy performed, which resulted in death. The brain was large, and presented the typical appearance of tuberous sclerosis, the nodules being found in both hemispheres, and one in each of the caudate nuclei. The ventricles were not distended, and their surface was smooth. The central canal of the cord was dilated. In the sclerotic areas the nerve cells and fibres were absent. In the neighborhood of the lesion they still existed, but appeared normal. Many of the cells were surrounded by large pericellular spaces. The lymphatic spaces were also distended, and the vessels were fewer than normal. The neuroglia appeared hyperplastic.

Case XXV.—Bourneville. The antecedents of the patient were distinctly neuropathic. Both the father and mother had had syphilis, and the latter had had numerous miscarriages. The first signs of mental deficiency were observed at the age of three months. At the age of thirty-three months a few words were spoken, and the patient made an effort to walk. Epileptic attacks commenced at the age of eight months, but soon ceased, and did not recur until the child was three years old. Death occurred at the age of eleven years. The pia mater was adherent to the cortex over the sclerotic nodules. These consisted of dense masses of neuroglia tissue, without nervous elements, although the neuroglia cells sometimes presented peculiar elongations, and contained fewer blood vessels than the normal tissue.

Case XXVI.—Tedeschi. The patient, a woman, lived to the age of twenty-eight years, and was married. She suffered from chronic epilepsy, and died from tuberculosis. The brain presented the characteristics of the disease. It was enlarged and firm, and about twenty nodules were

distributed upon the surface of the hemispheres. Upon section of the sclerotic nodules, they were found to consist of a gray peripheral zone, a pale, white, median zone, and an intensely red centre. Beneath the ependyma were a number of hard swellings, about the size of millet seeds. The sclerotic areas contained a few faintly staining varicose fibres. The neuroglia cells possessed threadlike processes, and formed a thick network, and some of them resembled ganglion cells, having large, pale nuclei and some branching processes. There was hyperplasia of the blood vessels, but no round-celled infiltration. The masses in the floor of the ventricles were composed of cells with long, delicate processes, that formed a network permeated by normal vessels. The ganglion cells of the thickened gyri were vacuolated, and sometimes the nucleus was deformed and near the periphery. In the other areas large cells, fifty-four microns in diameter, with large, pale nuclei, degenerated protoplasm, and branching processes were found, which Tedeschi holds to be ganglion cells similar to those found in glioma.

Case XXVII.—Spiller. There was no family history. The patient was an imbecile, and was epileptic; during his fits he would fall, striking the right parietal region of the head. The history is extremely meagre. The *ære* is not given. At the autopsy it was noted that both feet were in the equino-varus position. There were no contractures and only slight evidences of rachitis. The dura was not adherent. The brain was slightly *œdematous*, and contained a sclerotic area, about the size of a dollar, in the right frontal lobe. The cortex was narrowed. A similar area was found in the right parietal lobe. The other organs were normal.

Case XXVIII.—Jürgens. The family history was negative. Three weeks before delivery the mother had a slight fall. At the age of three months the child had a convulsion. This was repeated two weeks later. The child appeared, however, to have normal intelligence. Six weeks later general convulsions recommenced, and con-

tinued, with brief intervals, for several days. There was then paralysis of the right arm. The convulsions were repeated weekly, and were often associated with movements of the eyes, and preceded by a cry. The head was large, 41 cm., in circumference; the fontanelles were prominent and fluctuating. There was no disturbance of the reflexes and no contractures. Death occurred at the age of six months. At the autopsy the skull showed rachitic thickening; the dura mater was distended, but otherwise normal; the arachnoid and pia were thin and transparent; the convolutions were flattened, and many contained round, sharply circumscribed areas of a grayish white color and almost cartilaginous consistency. The upper surface of the lateral ventricles was rough, exhibiting here and there slight elevations, some almost as large as a cherry. The ependyma was thickened; sclerotic masses were also found in the basal ganglia. The spinal cord was normal, excepting the lower part of the lumbar region, which was slightly hardened. At the apex of the left ventricle there was a hard, spindle-form intumescence. Similar but smaller foci were scattered throughout the substance of the left ventricle, usually just beneath the pericardium. The mesenteric glands were enlarged; the kidneys contained cysts. Microscopical examination of the myocardial lesions showed sclerotic muscular fibres and areolar fibrous connective tissue, in the meshes of which were small, round or oval cysts resembling coccidia. Similar bodies were also found in the sclerotic areas in the brain. An emulsion of brain substance injected into three rabbits caused the death of two in two days without lesions, and of the third in eight days with typical parasitic myocarditis. Three rabbits were also inoculated in the eye, and one, at the time the paper was written, exhibited cerebrospinal symptoms.

Of these cases, the four reported by Buchholz and that of Henoch, should possibly be excluded as of some other nature.

In addition to these cases, similar pathological conditions are more or less definitely alluded to by other writers. Rokitansky speaks of the partial indurations of the brain, which are generally distinguished for the great degrees of hardness which they attain, and known as a callous state or cicatrix of the brain. These infiltrations, which he appears to regard as cancerous, sometimes render the diseased portion of the brain tough and firm, as if it were composed of fibrous tissue. According to Bourneville, Hayem, Hoffmann and D'Espine have referred to cases that may also be similar. Barlow and Bury also speak as if they had seen a number of examples, but give no details.

The common features of all these cases are, clinically, the existence of epilepsy with a greater or less impairment of the intelligence, and, pathologically, of a hyperplasia of the neuroglia, with the formation of tumorlike masses, and some destruction of the ganglion cells and nerve fibres. Hyperplasia of the neuroglia, without the formation of nodules, has also been described in epilepsy.

Chaslin examined three brains from epileptics, and found thickening and diffuse sclerosis of the cortex, that was due to a hyperplasia of the neuroglial cells and an increase of their fibres, which, he believes, may traverse the protoplasm of the cells, or be independent of them. The neuroglia seemed to invade, and, to a certain extent, to replace the surrounding structures, the most superficial layer of the cortex being chiefly affected. Often the nuclei of the neuroglial cells stained poorly, and their protoplasm showed a slight projection in one part. The ganglion cells were rare and irregularly arranged, and their protoplasmic processes altered. The vessels were usually normal, excepting in the sclerotic parts, where they were reduced in number and had thickened walls. The degree to which the process was advanced varied in different regions. Bleuler examined twenty-six brains obtained from chronic epileptics and idiots, in all of which

the neuroglia was increased between the tangential fibres of the cortex and the pia mater. This change was diffuse, but not uniform, in all parts of the brain, and its extent appeared to correspond more closely to the degree of idiocy than to the duration of the disease. The pia was not adherent. In fifty-four brains taken from patients not epileptic, there were slight indications of the sclerosis in fifteen (idiots, paranoiacs, paralytics and senile dements), but even in these cases the appearance was not the same; the fibres were not parallel and were more loosely arranged. Schultze found hyperplasia of the neuroglia in a case of porencephaly, probably congenital in nature. Corpora amylacea and compound granular cells were also found in the lesions, and there were warty prominences upon the floors of the ventricles. Pierrot has observed sclerosis in senile brains, affecting the deepest and most superficial layers of the cortex.

Various theories have been held regarding the nature of gliosis. Brissaud, in discussing the case which he reported with Bourneville, speaks of it as a chronic inflammatory process of slow development, with its chief seat in the gray matter. In the following year, in discussing the case of Pozzi, he stated that the only distinction between the hypertrophic and atrophic forms is that the former represents an earlier stage, and the latter a later stage of the process; that is, hypertrophic sclerosis represents hyperplasia of the neuroglia cells and fibres, atrophic sclerosis their subsequent contraction.

Hartdegen, however, whose patient died two days after birth, pointed out that the process must be congenital; but, as in his case there was no defect in the arrangement of the convolutions, he held that it must have commenced after the seventh month of foetal life, before which time the convolutions are not fully formed. Pollak is satisfied to call it disseminated sclerosis, which he believes may affect the foetus in utero, developing slowly or rapidly after birth. Fürstner and Stühlinger had a theory of their

own that to-day sounds somewhat absurd. They believed that the lymphocytes passed through the blood vessels and collected outside the adventitia, where they were converted into neuroglia cells, and that their gradual accumulation produced the sclerotic change. After this, pressure, or other cause, may produce softening in the centre of these areas; a process, they remark, and not unjustly, very similar to that which occurs in syringomyelia. They even claim to have observed these lymphocytes in the process of transformation. It should be noted that their specimens, all of which were from adults, showed distinct signs of inflammatory reaction. Chaslin, in 1889, believed that the sclerosis that occurs in epilepsy is due to a hyperplasia of the neuroglia, and proposed to call it neuroglia sclerosis. He points out that the neuroglia forms bundles of coarse fibres; an observation that had already been made by Buchholz, who explained it by supposing that the neuroglia cells were converted into fibres. In a paper published two years later he stated that he did not believe that the gliosis cerebri is due to an old inflammatory lesion, especially as the hyperplasia takes place at some distance from the blood vessels, and is most intense in the superficial layer, and nevertheless the pia is not adherent. He suggests three hypotheses: 1. Arrest of development of the nerve cells and fibres. 2. Only partial development of the nerve tissue. 3. Primary development of the neuroglia and atrophy of the nerve tissue by compression. He preferred the last, particularly as he regards the process as congenital, and as, in the regions where it was advanced, the capillaries are compressed. He concluded that: 1. The fibres in the cerebral cortex in sclerosis are developed from the neuroglia. 2. This process is found in epilepsy, is due to some congenital defect, and should be called gliosis. 3. In many conditions in which it occurs several causes are active.

Féré appears to include *sclerose tubereuse* among the chronic forms of encephalitis, although he admits that

Chaslin has demonstrated that it is due to proliferation of the neuroglia tissue and is peculiar to the nervous system.

Birch-Hirschfeld has suggested the possibility that the primary lesion is an atrophy of the nervous elements with consecutive proliferation of the neuroglia tissues. He is skeptical regarding the inflammatory nature of the process. Ziegler also rejects the inflammatory theory, and holds to that of congenital defect; although he states that the form known as ependymal sclerosis, that is, the thickening of the subependymal neuroglia and its projection into the ventricle, a condition commonly present in gliosis cerebri, is usually associated with degenerative or inflammatory conditions. Thibaut alone seems to consider nodular gliosis as a distinct disease, believing that there is a rapid hyperplasia of the neuroglia tissues, which presses upon the nervous elements and causes atrophy. A totally different view is taken by the English authors Barlow and Bury. They do not speak of tuberous sclerosis at all, but describe, among the changes occurring in the brain as a result of hereditary syphilis, conditions which are identical with it. The most striking and important lesions are found in the cortex. The sclerosis occurs in small masses, or may involve entire convolutions, with or without increase of their bulk. Sometimes there is a certain amount of atrophy, some of the convolutions being narrow, of cartilaginous consistency, and brownish pink in color. The white substance may be involved also, but ordinarily the change is entirely cortical. Microscopically there is found extensive overgrowth of the neuroglia and disappearance of the nerve cells; occasionally the membranes are adherent, and arterial changes are frequent, although not constant. Gowers, who appears to have devoted very little attention to this particular condition, adopts this view without hesitation. Hensch, although not suggesting any other etiology, does not believe that the cases are demonstrably

syphilitic, an opinion with which most pathologists will agree. Barlow and Bury do not describe any cases, fail to mention whether syphilitic antecedents usually are present, or if unquestionable syphilitic lesions are to be found in other parts of the body.

Examination of the recorded cases with reference to the etiology gives very unsatisfactory results. In the great majority it is not discussed. Of all the twenty-seven cases the sex is given in seventeen, six female and eleven males; the presence or absence of neuropathic heredity is mentioned in nine cases, and may be inferred in a tenth; it was present in six. Traumatism occurred in one case, and appeared to bear some etiological relation to the condition. Syphilis of the parents is mentioned in one case, and it may have existed in another. In a number of others it was apparently excluded on account of the existence of other healthy children in the family, both older and younger. It is interesting to note the condition of the kidneys. This has been recorded in only eight cases; in a number of the others they were certainly not examined, and in the rest no mention is made of either the presence or absence of changes. Of these eight cases, tumors of the kidneys, essentially similar in their gross appearance, were found in five, and the kidneys of a sixth case contained cysts. In one case congenital lesions of the heart were present. In a case of Von Recklinghausen, which may possibly belong to this condition, multiple myomata of the heart were found. The discovery of a coccidial parasite by Jürgens is of great interest; but, although it is apparently pathogenic for rabbits, it cannot be accepted as the cause of the brain lesions without further confirmation. Unfortunately, these cases appear to be so rare that this is not likely soon to be forthcoming. The congenital character of some of the cases and the entire absence of any bodies resembling coccidia from the lesions justify, for the present, a certain degree of conservative skepticism.

An examination of the lesions, with particular refer-

ence to the nature of the process, shows that the following features are more or less constant in all of the cases: The thickening of the neuroglia fibres and their arrangement either in wavy lines or in coarse mesh work. Freund describes it as a hyperplasia of the neuroglia, with increase of the cells, and atrophy and partial or total disappearance of the nervous elements. The neuroglia forms a coarse-meshed network, or bundles that may subsequently retract. This is found not only in the forms of sclerosis that appear to be idiopathic, but also as a change secondary to destructive injuries and infectious diseases, such as syphilis. Tedeschi and I have both found it as a result of experimental injury. Koppen has found it in a number of cases that were usually of syphilitic nature. In one the lesions were found in the occipital lobes, limited particularly to the cortex, and consisted of small areas somewhat lighter and denser than the ordinary tissue. These were of two kinds—those containing granular cells and fibrous connective tissue, and showing traces of nervous tissue, and those consisting of proliferated neuroglia, in which the glia cells were increased in number, and their fibres formed a wide-meshed network; in these areas there was an excessive number of blood vessels, and the ganglion cells and nerve fibres had disappeared. In a brain with microgyri, probably a case of atrophic sclerosis, the neuroglia in some situations was arranged in irregular fibrous masses, sometimes in wavy, sometimes in spiral form. There was no network, and neither glia cells nor nerve fibres could be found. In the subependymal tissue similar changes were present. Weygandt also observed a coarse reticulum in the neuroglia in the neighborhood of a gumma of the brain, and in some of the neighboring convolutions the cortex contained an excess of glia cells. I have observed similar changes in the cortex of a brain from a habitual criminal, which, excepting an apparent thinness of the gray matter, exhibited no gross lesions.

The pyramidal cells are usually entirely absent in the

most sclerotic foci; in other situations they are present, often apparently increased in number, and staining well, but they are atypical in arrangement, and show tortuosity of their protoplasmic processes, as occurred in my own case and in those of Hartdegen and Bruckner. The same condition has been described by Koster as occurring in the brain of an idiot who died at the age of twenty-six, and whose brain presented no gross lesions; by Bourneville and Pilliet, in a case of diffuse sclerosis of the brain and cord, and by Chaslin in his three epileptic brains. The pericellular spaces are occasionally distended. This, according to De Quervain, is not an *antemortem* condition, even although round cells are sometimes found in this space. Koster, however, looks upon it as a morbid lesion, having sometimes found two ganglion cells in one dilated space. It is certainly an artefact in many cases, however, and I do not personally believe that much weight can be attached to it. More important is the dilation of the perivascular spaces. Jendrassik and Marie were the first to call special attention to this. In their case this space was sometimes five or six times as wide as the vessel it surrounded, and was entirely occupied by a large-meshed reticular tissue, with cells at the intersections of the fibres that were not unlike neuroglial cells. The fibres either ceased at the inner surface of the lymphatic sheath, or they extended into the adventitia. The medullated nerve fibres stopped at the edge of the space as if cut off, and the vessels were tortuous and apparently more numerous than normal, both changes probably due to the retraction of the tissue. Muhr has described similar changes in a case of hemiatrophy of the brain. This distension of the perivascular lymph spaces seems to confirm Strümpell's theory of lymphatic obstruction.

The degree of vascularity has been variously described. Bourneville and Pilliet, and Tedeschi have found the blood vessels to be numerous, while Chaslin and Bourneville and Brissaud have stated that they are few, or absent from the

sclerotic areas. Buchholz regards some of the fibrous tissue in his case as representing degenerated blood vessels. In my case both conditions were present. Where the sclerosis was slight, the blood vessels did not seem to be increased in number. Where it was marked, particularly in those areas found in the deepest layers of the cortex, they were distinctly more numerous. In fact, it sometimes appeared as if an agiomatous alteration had taken place. In the subependymal nodules, on the other hand, where the changes were by far the most pronounced, the blood vessels were entirely absent. It seems easy to understand how these variations occur. The proliferated neuroglia tissue must exert more or less pressure upon the surrounding tissue. In certain areas this pressure causes obstruction, with secondary dilatation of the blood vessels, and, perhaps, the approximation of certain adjacent branches. That an actual vascular proliferation takes place it is impossible to deny, but there seems to be no good reason to believe that it does, unless we accept the theory of Birch-Hirschfeld that there is a primary atrophy of the true nervous elements and secondary proliferation of the other tissues to take their place; but this theory appears to be disproved by the frequently reported presence of pyramidal cells and nerve fibres in those areas where the sclerosis has not reached an extreme degree. The ultimate disappearance of the blood vessels is probably due to their total obliteration by pressure. Comparing two neighboring nodules I saw numerous vessels in one, while in the other, in which the neuroglia fibres were coarser and the process appeared to be more advanced, they were totally absent. Perhaps the peculiar bodies found in the sclerotic areas beneath the ependyma of the lateral ventricles are more significant of the destruction of nerve tissue. Pozzi describes them, and calls them colloid, and they are probably the same as the round, glassy bodies that Bruckner mentions. Dagonet has described what are evidently the same things, and calls them

hyaloid. He believes that they are derived from cerebrin. Stroebe found them in the cords of rabbits, in which partial or total section had been made, and from their relation to the nerve fibres is convinced that they are the products of the degeneration of the myelin sheaths and the axis cylinders. They have been frequently described in degenerative processes, and I have observed them in an area of softening in the brain of an infant that died of tubercular meningitis, and in the sclerotic areas in the cortex of the brain of a criminal. The peculiar pale, pear-shaped bodies found in the cortex are more difficult to explain. Bourneville, Tedeschi and De Quervain have found similar bodies and considered them as degenerated cells, either neuroglia or ganglion, an opinion with which I agree. If we consider them to be neuroglia cells, they suffice to explain the disappearance of these from the areas where the gliosis is most pronounced. In all the positive cases endarteritis and perivascular round-cell infiltration appear to have been absent.

It does not appear from this analysis that there is any ground for the assumption of either an inflammatory or syphilitic etiology. Either process can produce hyperplasia of the neuroglia tissue, but the absence of the round cells and proliferation of the true connective tissue, and the infrequency of pial adhesions are sufficient to exclude the former. Syphilis produces such a variety of lesions that it is difficult to state positively that it could not cause nodular gliosis. Von Bechterew, however, holds that syphilis of the nervous system is a disease of specific nature, caused directly by luetic infection, and not a condition which may develop as a result of preëxisting syphilis. In the case of disseminated syphilitic sclerosis that he reports, the focal lesions appear to have been due to proliferation of the perivascular connective tissue, and not to hyperplasia of the neuroglia, and there was extensive endarteritis. The inclusion of pure gliosis among the syphilitic conditions by Barlow and Bury seems wholly without

warrant. The clinical histories are also negative; the fact that the parents of one child were certainly syphilitic is offset by the fact that in several cases we can be reasonably certain that they were not. The case of Henoch, in all likelihood, does not belong to this group; for in none of the others do the nodules bear the slightest resemblance to gummata. We are then forced to fall back upon some theory of the idiopathic proliferation of the neuroglia. In this connection it is interesting to note the considerable number of congenital defects that have been reported as occurring in these cases. In eight cases in which complete autopsies were made, tumors or cysts of the kidneys of considerable size were found in six. Unfortunately, satisfactory diagnoses have not been made in any of these cases with the exception of my own, although Bourneville speaks of one of his specimens as being cancerous, and Henoch of the nodules in his case as gummata. As the microscopic description of two of them at least agrees closely with the condition I observed, I think it not unlikely they were tumors similar to mine, that is, adenosarcomata of a distinctly congenital type. In no case except my own, as far as I am aware, were nodules found in any other part of the body. It seems to me therefore reasonable to conclude that hypertrophic tuberous gliosis is akin to tumor formation, and represents a growth peculiar to late foetal existence in view of observations of Binswager and Hartdegen. It is closely allied to atrophic sclerosis, a condition most carefully described by Jendrassik and Marie, and to sclerosis with cavity formation. These varieties are not sharply separated, but any two, or all three, may co-exist in the same brain. Bourneville has reported one case and Fürstner and Stühlinger two, in which cavities were found in sclerotic nodules, and in nearly all cases of the hypertrophic form some of the convolutions were atrophic.

The symptoms of this disease are exceedingly various. The essential ones are epilepsy, commencing in some form or other in early infancy, associated with a greater or less

degree of idiocy. Richardière holds that in the hypertrophic form the convulsions are more frequent and severe, and the idiocy more profound, than in the atrophic form, but this is doubtful. It is said of Schule's case that she learned to read and write, although she was always unable to reckon; and Bruckner's patient learned to write a little. Tedeschi's subject was a married woman of 28. Pozzi's, an old man of 63, who had been able to keep out of an asylum for that many years. My own case exhibited remarkable skill in untying knots, no matter how complicated. The epilepsy also may be reduced to transient attacks of rigidity, as in Pollak's patient, or completely absent for long periods, as in Bruckner's case. The epileptic attacks, as far as one can gather from the often meagre reports, are sometimes Jacksonian in type, sometimes more general. The other symptoms, such as nystagmus, muscular atrophies, facial atrophy, are too infrequent to be seriously considered in making a diagnosis; difficulty of deglutition has been noted in Simon's and Pollak's cases, and in my own. The value of symptoms indicating focal lesions is doubtful. When the very considerable microscopic alteration of the cortex and the fact that the lesions are often found in the motor and visual regions are considered, it must be admitted that corresponding functional disturbances cannot exist. This is not improbable, in view of the frequent presence of the ganglion cells in the sclerotic areas of the cortex. Bourneville holds the clinical diagnosis to be exceedingly difficult. Freund, that it is impossible, claiming that the disease cannot be differentiated from diffuse atrophic sclerosis, or meningo-encephalitis; but Simon confidently describes the clinical course, and reports a number of living cases, as does also Richardière, although the latter admits the uncertainty of his diagnoses. Bruckner, in his own case, ascribes the idiocy to the involvement of the frontal lobes, and the epilepsy to the lesions in the motor region; but Simon's patient had no lesions in the frontal lobes, and

was nevertheless an idiot, although it must be admitted that a certain degree of microcephaly might account for this. It is not unlikely, however, that the process, even in the hypertrophic nodular variety, is tolerably diffuse; for I have observed slight changes in portions of the cortex that are apparently normal. In my own case, the greater extent of the process on the left side may have been associated with the invariable tendency to fall to the right. Unfortunately, no accurate record of the condition of the eyes was kept, otherwise it is not impossible that the symmetrical lesions in the cuneus might have been suspected.

The following conclusions seem to be warranted:

1. There is a morbid process characterized by a hyperplasia of the neuroglia cells and fibres that leads to gradual atrophy of the nerve fibres and the ganglion cells, and is associated with perivascular changes of doubtful nature.

2. The first manifestations occur in early life, often a few weeks after birth, and anomalies or congenital tumors are sometimes found in the same cases.

3. All cases of this disease are epileptics, and many of them idiots.

4. The cause is unknown, but the disease probably commences before birth and after the seventh month of foetal existence, and is of the same nature as gliomatosis.

Scarpatetti (*Archiv für Psychiatrie*, vol. 30, No. 2, p. 537) has recently reported an additional case of this nature. The father of the patient was unknown; the mother had always been sickly. The child had visited school in her youth and had learned something. In later life she had been at service, and had had three illegitimate children. The first is still living, the second died of hydrocephalus, the third she strangled. The investigation instituted by the court developed that she had suffered for years with occasional attacks of epilepsy. She was small, undeveloped and stupid. Her head had a circumference of 58 cm. There was a dermoid cyst on the left side of the nose; the ears were especially small, and there

was torus palatinus. The patient was industrious, but appeared to take no interest in anything but her manual work. At the age of 20 she was attacked with fever, diarrhoea, pains in the legs, and paraplegia, dying on the third day of the attack. At the autopsy the skull was found to be thick and solid, the dura slightly adherent. In the substance of the brain were four hard, tumorlike masses, between the first and second frontal lobes. Many of the convolutions were pale and of the consistency of cartilage. The larger swellings were depressed in the centre, and over them the pia mater was adherent. Numerous small, hard nodules were found in the ependyma of the ventricles. The cerebellum, basal ganglia and spinal cord were normal; the kidneys contained a number of small, hard, circumscribed nodes, consisting chiefly of involuntary muscle fibres. Microscopically, no degenerated fibres were found in the sclerotic areas. The pyramidal cells were either slightly swollen or atrophic. They were much altered in shape, and their position was irregular. The tangential fibres had disappeared, and at the periphery of the cortex nothing was left excepting a coarse neuroglia tissue. The other regions in the brain were normal. The blood vessels exhibited marked hyalin degeneration, and were in part obliterated. In the sclerotic areas they were more numerous than normal. The author calls attention to the presence of other signs of degeneration, but is rather inclined to believe that the disease is the result of hereditary syphilis, and, on account of the normal position of the chief sulci, he is inclined to believe that it developed late foetal life. He admits, however, that it probably is only an exaggeration of the condition usually found in epileptic brains.

Scarpatetti refers to an additional case, that, on account of its misleading title, escaped my attention, and has not been included in the literature. The changes were apparently slight. (Koch: "Ein Fall von Idiotie in Folge von Application der Zunge." *Neurologisches Centralblatt*, 1887.)

No.	Name.	Age at death.	Sex.	Neuropathic heredity.	Syphilis.	Tumors of the kidneys.	Trauma.	Congenital defects.
1	Bourneville.	15	f.	0		I		
2	Bourneville & Brissaud	4	m.	I				I
3	Bourneville & Bonnaire	5	m.	I		I	I	
4	Bourneville & Bonnaire	5	m.	I	?	I		
5	Bourneville & Bonnaire	5 $\frac{3}{4}$	m.	I		I		
6	Hartdegen.	2 days	m.	0		0		I
7	Pollak	3	f.	?				
8	Bruckner.	22	f.	0				
9	Pocci.	68	m.					
10	Simon.	2 $\frac{1}{2}$	f.	I				
11	Schule							
12	Bucholz.							
13	Thibal.		m.					
14	Bucholz.		m.					
15	Bucholz.		m.					
16	Bucholz.		f.					
17	Wilmarth.							
18	Wilmarth.							
19	Wilmarth.							
20	Wilmarth.							
21	Wilmarth.							
22	Wilmarth.							
23	Henoch.	2			?	I		I
24	Berdez.	4	m.	0.	?			
25	Bourneville.	11	m.	I	I			
26	Tedeschi.	28	f.					
27	Spiller.		m.			0		
28	Jurgens.	6 mos.	m.	0	0	I	?	I
29	Sailer.	15	m.	I		I		
30	Scarpattetti.	20	f.	I	?	I		I

I=yes. 0=no.

LITERATURE.

- Barlow & Bury: Tuke's Dictionary of Psychological Medicine, London, 1892, p. 1261.
- Von Bechterew: Arch. für Psychiatrie, vol. xxviii., No. 3, 1896.
- Berdez: Ziegler's Beiträge, vol. xvii., p. 648.
- Binswanger: Virchow's Archiv, 102, 1886.
- Birch-Hirschfeld: Lehrbuch der pathologischen Anatomie, vol. ii., Leipsic, 1894.
- Bleuler: Münch. med. Woch., Aug. 15, 1895.
- Bourneville: Arch. de Neurologie, 1880, p. 91.
- Bourneville: Progrès Médical, 1896, No. 9.
- Bourneville et Bonnaire: Bull. de la Soc. Anat. de Paris, Feb., 1881.
- Bourneville et Bonnaire: Bull. de la Soc. Anat. de Paris, July, 1881.
- Bourneville et Bonnaire: Progrès Médical, 1881, p. 1007.
- Bourneville et Brissaud: Arch. de Neurologie, 1880, p. 397.
- Bourneville et Pillet: Recherches sur l'Epilepsie, l'Hystérie et "Idiotie par Bourneville et Bricon, Paris, 1885.

- Bruckner: Arch. für Psychiatrie, vol. xii., No. 3, 1882.
 Buchholz: Arch. für Psychiatrie, vol. xix., 1888, p. 591.
 Buchholz: Neurologisches Centralblatt, 1890, p. 636.
 Chaslin: Semaine Médicale, 1889.
 Chaslin: Arch. de Medicine Experimentale, vol. iii., 1891.
 Dagonet: Système Nerveux Central, Paris, 1897.
 De Quervain: Virchow's Archiv, 133, 1893.
 Echeverria: Epilepsy, New York, 1870.
 Féré: Traité Elementaire d'Anatomie Médicale, Système Nerveuse, Paris, 1891.
 Freund: Infantile Cerebrallähmungen, Vienna, 1897.
 Fürstner und Stühlinger: Arch. für Psychiatrie, vol. xvii., 1896.
 Gowers: Diseases of the Nervous System, Philadelphia, 1896.
 Hartdegen: Arch. für Psychiatrie, vol. xi., 1881, p. 117.
 Henoch: Kinderkrankheiten, Berlin, 1893.
 Jendrassik et Marie: Arch. de Physiologie Normale et Pathologique, 1895.
 Jürgens: Berl. klin. Woch., April 4, 1898.
 Koppen: Arch. für Psychiatrie, vol. xxviii., No. 3, 1896.
 Koster: Neurologisches Centralblatt, 1889, p. 292.
 Muhr: Arch. für Psychiatrie, 1876, p. 733.
 Pierret: Bride. Thèse de Lyon, 1888 (quoted by Féré).
 Pollack: Arch. für Psychiatrie, 1881-2.
 Pollak: Deutsches Arch. für klin. Med., vol. xxiv., 1879, p. 407.
 Poszi: L'Encephale, 1883, p. 157.
 Richardière: Thèse de Paris, 1896.
 Rokitsky: Manual of Pathological Anatomy, London, 1850.
 Sailer: Proceedings of the Pathological Society of Philadelphia, vol. i., No. 3.
 Schule: Klinische Psychiatrie, 3d edition, 1886.
 Schulze: Festschrift, Heidelberg, 1886.
 Stroebe: Ziegler's Beiträge, vol. xv.
 Simon: Rev. Mens. des Maladies de L'Enfance, Dec. 1883.
 Spiller: Reports of the Philadelphia Pathological Society, 1896-7.
 Strümpell: Arch. für Psychiatrie, vol. ix., p. 268.
 Tedeschi: Revista Sperimentale di Freniatria e di Med. Legal, vol. xx., 1894, p. 332.
 Tedeschi: Ziegler's Beiträge, vol. xxi., No. 1, 1897.
 Thibal: Thèse de Paris, 1888 (quoted by Chaslin).
 Weygandt: Arch. für Psychiatrie, vol. xxviii., No. 2, 1896.
 Wilmarth: Alienist and Neurologist, 1890.
 Ziegler: Lehrbuch der pathologischen Anatomie, vol. ii., Jena, 1895.

A CASE OF SEROUS (ALCOHOLIC) MENINGITIS SIMULATING BRAIN TUMOR.¹

By THEODORE DILLER, M. D.,

Neurologist to the Allegheny General Hospital; Visiting Physician to the Insane
Department of St. Francis Hospital, Pittsburg.

The case of serous meningitis to be related was mistaken by me for one of brain tumor. The symptoms presented by it were so strikingly like those produced by an intracranial neoplasm, that I am led to hope that a brief account of the case may be of some value.

Mr. X., aged 33, formerly a strong, vigorous man, was seen by me on March 5th, 1898. For many months he had been indulging in large amounts of alcoholic drinks.

He had been, for many years, subject to sick headaches and vomiting attacks. For two or three months back these headaches had become very much more severe in character and longer in duration, and largely localized in the frontal region. The patient had latterly spoken of them as head pains rather than headaches. They sometimes lasted two or three days at a time, and were constantly growing more and more severe.

During the previous six months vomiting spells had grown more frequent. They occurred at any time in the day, and, indeed, they often came upon him in the night. They were accompanied by little or no nausea, and occurred whether the stomach was full or empty. Since the increase in the frequency of the vomiting attacks he had constantly grown thinner.

For about five months his eyesight had been steadily failing. Three months ago it was noted that he staggered in walking, and as time went on this became more and more apparent. For two months past he had complained of pains in the thighs and legs, chiefly in the latter, and of burning in the soles of the feet.

On February 23d, 1898, ten days before I saw him, he was seized, for the first time, with a convulsion. It was general in

¹ Presented at the annual meeting of the American Neurological Association, held in New York, May 26th, 27th and 28th, 1898.

character, accompanied by unconsciousness, and lasted only a few minutes. An hour later he had a similar convulsion, and a third about an hour after this. For two weeks preceding these convulsions the headaches had been especially agonizing.

Since the convulsions he had become possessed of many fleeting delusions with regard to time, place and persons, some of which were of a painful character. His memory was defective, and he was in a condition of more or less semi-stupor.

This was his mental condition when I first saw him. In conducting my examination I had little difficulty in getting him to do as I wished. But after I left he remarked that he guessed that B—— was trying to act "smart," being under the impression that I was the local confectioner, with whom he was well acquainted.

He staggered in walking, and his gait was one of weakness. He was considerably emaciated; his muscles were flabby. There was a good deal of tenderness to pressure in the legs and feet, and a moderate amount in the thighs. Both knee-jerks were much diminished. No ocular palsies were present. The pupils reacted to light and in accommodation. He was unable to distinguish various coins, or the large letters of newspapers. There were no cranial nerve palsies. The urine showed no albumin or sugar. His temperature, from the time I saw him up to his death, varied between 99 and 100. Double optic neuritis was present.

The patient gradually sank into coma, and died six days later while in that condition.

The autopsy failed to reveal a brain tumor, but disclosed great dilatation of the pial vessels, with a large amount of clear, colorless fluid beneath the pia. There was intense congestion of the pia in several spots. But little more than the usual amount of fluids was found in the ventricles.

To briefly recapitulate. The patient had been ailing and going steadily downward for six months, presenting during this time the following symptoms: Severe headaches; frequent vomiting attacks; failing vision, due to optic neuritis; weak, ataxic gait; loss of knee-jerks; tenderness in the legs and burning in the feet, and, finally, general convulsions followed by marked mental changes.

It seemed quite plain that the man was suffering from multiple neuritis, due to alcoholism. The ataxic gait, weakness, wasting and tenderness of the legs and burning of the feet, with diminished knee-jerks, were explained

very reasonably by this diagnosis. There remained, however, the headaches, vomiting, optic neuritis, convulsions and mental symptoms to be accounted for. These seemed to me to indicate the presence of a brain tumor, although I recognized the fact that the mental symptoms were very like those seen in alcoholic multiple neuritis. Indeed, I was uncertain how much these symptoms should be attributed to the supposed brain tumor, and how much to alcoholism.

While nearly all the cardinal general symptoms resulting from brain tumor were present in this case, there were two features which should, perhaps, have led one to recognize the real situation, viz., the history of alcoholism and the presence of multiple neuritis. The diagnosis of brain tumor and multiple neuritis assumed a coincidence which would have been unnecessary, had the true condition been recognized.

Acute serous alcoholic meningitis, as described, for example, by Dana,¹ could not easily be mistaken for a brain tumor, but it is in chronic cases of this disease that there is, as has been so well pointed out by Quincke² frequently a great liability of this mistake being made. Oppenheim,³ indeed, calls attention to the striking resemblance of the symptoms produced by chronic serous meningitis and those produced by brain tumor, and further says that in most cases of the first-named disease the diagnosis of brain tumor has been made. In this latter statement of Oppenheim I find a measure of personal comfort.

¹ Medical Record, vol. lii., p. 801, et seq.

² Sammlung klinischer Vorträge, 1893, No. 67, p. 655.

³ Berliner klin. Wochenschrift, Nos. 49 and 50.

NEW PATHS IN PSYCHIATRY.¹

(INAUGURAL ADDRESS) BY FREDERICK PETERSON, M.D.

President of the New York Neurological Society.

Eighty years ago a medical student engaged in the commonplace work of walking the London hospitals, with a young mind open to thrilling impressions, came unexpectedly upon a new domain, not a domain of medicine, except as that is related to and bounded by the vast domains of human knowledge, but a "wide expanse" of fancy and imagination, the discovery of which led him to exclaim:

"Then felt I like some watcher of the skies
When a new planet swims into his ken;
Or like stout Cortez when with eagle eyes
He stared at the Pacific—and all his men
Looked at each other with a wild surmise—
Silent, upon a peak in Darien."

Thus we, the voyagers of a later day, in an older period of the world's history, with our several conquests of the secrets of Nature behind us, stand upon the threshold of unexplored regions, and may well regard each other "with a wild surmise."

A writer in a recent number of the *Revue Scientifique* describes Psychiatry as the new Prometheus which shall wrest from Nature the secrets of thought.

All over this earth, in a hundred clinics and in a hundred laboratories, patient investigation is being carried on. Man is studying the biological unit of himself. What a problem that unit is! What an array of specialists is engaged in the work! There are the anatomist, the comparative anatomist, the histologist, the comparative histologist, the cytologist, the comparative cytologist, the

¹ Read before the New York Neurological Society, May 3, 1898.

physiologist, the comparative physiologist, the chemist, the embryologist, the ethnologist, the pathologist, and many more, who busy themselves with the body. There are the psychologist, the comparative psychologist, the psychiatrist, the criminologist, the philologist, the pedagogue, and the innumerable others, whose labors are with the mind of man. Almost every one of these particular fields of work is subdivided into still other specialties, so that it requires even now a singularly broad and all-embracing mind to collect, digest and build up the facts already accumulated into some structure of great truth or general principle.

We who are students of the phenomena presented by the normal and by the disordered nervous system, while we realize the enormous progress made in our province during the last twenty years, in localization of functions, in the pathogeny of nervous diseases, in discoveries like that of the causation of cretinism and myxœdema, in the understanding of the minute structures and relations of centres and tracts, in the investigation of the laws of heredity, in the study of degeneracy and its stigmata, in the knowledge of the development of the mind from infancy to adult life, in the realms of hypnotism, hysteria, aphasia, and the like, while we realize all this, we still feel that we stand but in the half-light of discovery, and that there extend far out before us innumerable pathways leading into unknown regions, wherein shines the dim and fitful light of new truths to be attained:

"As, in one eye,
Light, from unnumbered worlds and furthest planets
Of the star-crowded universe, is gathered
Into one ray."

Thus spake another English medical poet,² who sixty years ago practised medicine in Zurich, and busied himself with translating "Grainger on the Spinal Cord" into German.

² Thomas L. Beddoes, author of *Death's Jest-Book*.

One of the most bewildering, intricate and mysterious of these pathways is that of physiological and pathological chemistry. But the explorers who follow this trail must need be "invincible athletes" if they shall penetrate far into the labyrinth of disturbed chemical metabolism in the numerous tissues of the body, and bring us practical facts in regard to the disordered functions, auto-intoxications, etc., produced thereby.

The roads of normal and pathological histology and cytology are more worn and better followed, but their many travelers are still remote from any final bourne.

It is not the happy privilege of many of us to be able to enter upon researches of these great proportions. Time, strength and means are not adequate for all of us to study the body and mind of man as a biological unit. The greatest discoveries, the most far-reaching results, must be attained by aggregations of specialists trained in many departments. These are now and then fortuitously associated in contributions scattered in different periodicals or monographs in many tongues. But far more satisfactory will be their work in so promising an aggregation as has recently been established in this city by the Commission in Lunacy of the State of New York, under the management and inspiration of Van Gieson, and in the well-equipped private laboratories of one or two of the members of this society, and in the several combined laboratories of foreign psychiatric clinics.

Yet, though some of these paths may not be open to us all, there is at least one of the roadways leading into the realms of the mind which any one of us may follow. It lies in the direction of the better clinical examination of our cases from the standpoint of psychology.

The neurologist may garner a vast number of extremely valuable data by the application of some of the principles and apparatus of the new physiological and experimental psychology to the investigation of his cases of organic brain disease. These patients have rarely, if ever,

been carefully studied in relation to their mental phenomena. We are only beginning to appreciate, for instance, the utility of the psychological examination in lesions of the frontal lobes. But I believe it to be of the greatest importance to study the quality, intensity and tone of sensations, the contents, distinctness, energy and emotional character of ideas, the evolution, durability and associations of ideas, the disorders of the affective life in the way of depression, exaltation, irritability, apathy and mutability, the disorders of the idea-association in the way of memory, attention, accelerated or retarded flow, coherence and ethical feeling, and, finally, the disorders of the judgment associations in the way of falsification or defect, in all of our cases of organic cerebral disease, in tumors, hemorrhage, softening, multiple sclerosis or other lesions, and even in diseases of the cerebellum. We cannot yet tell what lacunæ may not be thereby discovered in the psychic unity of the affected individual. Our studies of aphasia have been remarkably deficient as regards their psychic side.

In the investigation of the functional disorders of the brain also, there are fine conquests to be made by means of recent psychological methods.

While the neurologist has much to gain by following the psychological path in the study of neurological cases, far more vast is the expanse that opens out to physicians in reformatories, prisons, institutions for idiots and asylums for the insane, if they will travel the new road, under the new guidance, in the dawn of the new day!

When I look back upon three years spent in asylum work without light or guide, it seems to me that, aside from some practical gain in methods of management of patients and a certain familiarity with types of insanity acquired, I traversed a somewhat barren waste. It would be an inestimable privilege to live again through such opportunities, to be awake and not asleep, no longer benumbed by the slumbrous psychiatric dissertations of that day.

Doubtless these new psychological theories will have their period, and give place, in turn, to other and better ones, but there is a pleasant fascination in regarding mental phenomena by scientific method, a certain charm in sifting the psychical processes down to stimulus, sensation, idea-association, movement; in which series the emotions have a place only as attributes or properties of sensation and idea, while voluntary action is merely the result of a play or battle of motives in the idea-association (*Ziehen*). With some such scheme of investigation before us, it becomes a delight to study the disorders of sensation, the disorders of the memory-pictures or ideas, the disturbances of the idea-associations, the derangement of the judgment-associations, the influences of these various disorders upon the movements, action or conduct, the affections of the emotional tone of sensations and ideas, and, finally, the whole character of the individual as made up of the sum of the specific ethical feelings that are associated with his more complicated ideas.

But this is not the occasion, nor is there time, to give more than a passing glance at the rich region that lies with limitless horizon before every physician who has to do with morbid minds in his private practice, or in special institutions, if he but follow the new paths.

Ever since I observed the splendid facilities for study in some of the foreign psychiatric clinics, it has seemed to me a misfortune that not one of our large cities on this side of the water is provided with such a centre for psychological investigation. The psychological laboratories attached to some of our universities, dealing, as they do, with the normal mind, can never hope to accomplish as much in the way of new discoveries as similar foundations associated with clinics for nervous diseases or asylums for the insane, where is gathered together an abundant morbid material upon which to draw for the solution of many a psychic riddle. For it is true that most of our knowledge of normal functions of the human body, physiological or

psychological, has been gained by the physician through investigations conducted when these functions were perverted or destroyed by disease.

Surely this great city, blessed with so many hospitals, charities and institutions of learning, possessed of so many citizens eager to employ their large wealth for benevolent purposes and for human progress, might well lead the cities of the New World in the establishment of a psychopathic hospital, a psychiatric clinic, fully equipped with all adjuncts for clinical, chemical, psychological and pathological investigation. A psychopathic hospital would accomplish great practical good. It would be a boon to the many insane now gathered daily into a pavilion at one of our hospitals merely for distribution to various asylums. In such a hospital many cases could be treated and cured, thus avoiding transfer and commitment to asylums. Medical students and special students of psychiatry would profit from the convenience of access to the psychiatric clinics, and the young graduate would enter upon practice with some definite knowledge of insanity and its treatment. But the greatest value of the proposed special hospital would undoubtedly be the opportunities afforded for those aggregate studies by many specialists, which are destined one day to discover the origin and cure of many of the psychoses, and incidentally to unravel some of the mysteries of mind.

Clinical Cases.

A CASE OF UNIVERSAL MUSCULAR ATROPHY.

By H. A. HARE, M.D.,

Professor of Therapeutics in the Jefferson Medical College.

The patient that I show you has the following history:

Mrs. E. K., aged 45, married, a housekeeper, was admitted to the Jefferson Hospital, March 1st, 1898. She was born in Dublin, Ireland.

FAMILY HISTORY:—Her father died at the age of fifty of unknown cause. Her mother is living, is in good health, and is sixty-three years old. She had two brothers, both of whom died in infancy from unknown causes, but she had no sisters. There is no history of any hereditary disease.

PERSONAL HISTORY:—She contracted measles and whooping-cough when a child, and had a severe attack of mumps seven years ago, which she contracted from her children. She had malaria in 1882, but has never been seriously ill until her present trouble developed. In 1872 she was married, and has had ten children, all of whom are living and well, except one, which died of croup when five years of age. She has had no miscarriages or uterine trouble.

PRESENT ILLNESS:—Her last child was born in July, 1893. For about one month previous to its birth she suffered with a great deal of pain in the lower extremities, especially in the knees and feet, and the feet became swollen and dropsical. After confinement these symptoms disappeared, and she had no further trouble until three years ago, when the condition gradually returned, the lower limbs, especially the knees and feet, becoming painful and the feet being swollen. This condition persisted, but did not become serious until one year ago, when she was obliged to take to her bed, to which she has been confined ever since. During the last year her hips, shoulders, elbows and hands have been implicated, and she has gradually lost the use of the joints, the tendons becoming contracted and the joints deformed. The deformity is particularly marked in the hands, the right especially presenting the typical "seal-fin" hand appearance. She has lost a great deal of weight during the past year, and is weak and emaciated. She is unable to walk, and attributes this more to weakness than to deformity. The knees are sharply flexed, and she is



Universal Muscular Atrophy.

not able to straighten the limbs. The toes are sore and tender to touch. For the last six months she has had an intermittent vaginal discharge, dark in color, and of an offensive odor, not large in amount, but the gynæcologist reports no serious uterine lesion. The menopause began in the summer of 1896. Small bed sores are present over the sacrum, apex of left scapula, and on the left elbow. Her appetite is good, and her bowels are regular. The examination of the eyes shows that the color fields are reversed, and the visual fields greatly contracted. The urinary examination is negative.

As you will see from an examination of the patient's limbs and trunk, she is the subject of universal muscular wasting of a rather unusual type. You will also notice that an examination of her eyes reveals reversal of her color fields and marked limitation of her fields of vision, and her facies is distinctly hysterical. While at first glance the seal-finch hands and the bent limbs remind one of the posture of a person suffering from arthritis deformans, a careful examination of the joints shows that there is no marked disease of their surfaces nor the development of exostoses locking the joints. On the contrary, the deformities are chiefly due to muscular contractures. The difficulty in extending her limbs depends upon the condition of contracture in the muscles of the arms and thighs. Quiet, constant extension of a limb, kept up until the muscles are tired, causes stretching of these muscles, so that both the arms and legs can be almost completely straightened out.

I bring this case before you, not only in order to exhibit it, but with the hope that the members present will express their views as to the correct diagnosis to be reached.

When I first saw her I concluded that it was a case of rheumatoid arthritis, with secondary muscular wasting, complicated with distinct hysterical manifestations, which might be in part responsible for the muscular contractures, and this diagnosis I am still inclined to adhere to. The fact that the muscles of the trunk are as much wasted as those of the extremities is, however, an interesting fact to be considered.

CASES OF OPHTHALMOPLEGIA.

(From the Department of Prof. B. Sachs at the New York Polyclinic.)

I. A CASE OF FUNCTIONAL (HYSTERICAL) OPHTHALMOPLEGIA. REPORTED BY B. SACHS, M. D.

Functional ophthalmoplegia is sufficiently rare to warrant a brief report. The patient—F. S.—was kindly referred to me by Prof. Marple, to whom I am indebted for a careful ophthalmoscopic examination, and for the charts of the visual fields.

The patient (Fig. 1) is 51 years of age; for many years he has been a heavy drinker of beer; also gives an uncertain history of previous specific infection. He has been married during the past 27 years; his first wife, who is said to have had a *left ocular palsy*, died from a paralytic stroke about seven years ago. Six years ago he married his second wife. The patient led a regular life; attended to his business as a liquor dealer; at one time he weighed 300 pounds; now weighs 230 pounds. He was in good health until January 12th, 1898. On that day he attended the funeral of a friend. While standing on the sidewalk, waiting for the coffin to be carried out, he noticed a flash of light before his eyes, began to see double, felt slightly dizzy, and could not look into the light. At no time did he lose consciousness. He was badly frightened; knew that he had the same trouble from which his first wife suffered, and of which she died. Some friends assisted him to his home; on his arrival he found that both eyelids drooped; the photophobia was intense, and this symptom has been the most distressing one ever since. At the time of my first examination (March 5th, 1898), the following record was made: Double ptosis; when asked to look up makes a great but unavailing effort; slowly raises the entire head,

but does not use the *frontales muscles*, as patients with ptosis ordinarily do. After repeated commands, manages to raise the eyelids a little, but does not do so again, though requested to do so frequently, during the course of a long examination. On lifting the eyelids with the fingers, it is seen that the axes converge slightly. Conjugate movement of the eyes to the left is imperfect; to the right the movement is performed easily. Upward and downward movements of both eyes are slightly limited and done in a jerky manner. Testing the eyes singly, it is noticed that the l. rectus externus moves the eye around about half its normal distance. All other movements are performed more satisfactorily; but the movements are jerky, and at times succeed very much better than at others; muscles appear to be easily fatigued. The pupils are slightly irregular in contour; they react, though feebly, to light, but contract promptly during accommodation. Dr. Marple reported that there are no fundus changes. There is some apparent contraction of the visual fields. He has no central color scotoma, and his visual fields for color show no marked peculiarity. He has some choroidal changes, such as are usual in myopes; he also has some astigmatism.

Left vision is 20/40; with —1D. cyl. ax. 90=20/30.

Left vision is 20/40; with —1D. cyl. ax. 90=20/30".

A complete examination of the patient did not reveal any palsy in any other part of the body; the reflexes were normal, and there was no ataxia. Gait and station were normal. The head was held in a rigid position, with a slight curve to the right, from an evident desire to avoid the light. The only other symptom was a *complete left hemianalgesia*, with the exception of the left cornea, which was sensitive to touch; in every other part of the left half of the body, including the left nostril and the left half of the tongue, severe pricks with a pin were not felt. Touch, temperature and muscular sensations were normal in both halves. In the further progress of the case (to May 1st,



FIG. I (Case I).



FIG. II (Case II).

1898) few changes were observed until after an attempt had been made to affect the trouble by hypnotic suggestion; since that time improvement has set in, but the analgesia remains as before, and no effort has been made to remove the same by suggestion.

The interpretation of the symptoms was not altogether easy. In view of the alcoholic and (possibly?) specific history of the patient, it was natural to think first of an organic lesion; but the sudden origin of the palsies, the unusual variability of the symptoms during a single examination, the incomplete palsy of each muscle affected, the palsy of movement rather than of individual muscles, the preservation of the pupillary reflexes, the normal functions of the chief muscles supplied by the third nerve, in spite of the ptosis, militated against the diagnosis of an organic ophthalmoplegia. Moreover, no organic lesion could possibly account for the intense photophobia and for the contraction of the visual fields. On the other hand, the sudden onset amid emotional excitement (the burial of a friend, and the recollection of his wife's similar trouble), the variability of the symptoms, and, above all, the hemianalgesia, not to mention the improvement under hypnotic suggestion, support the diagnosis of a functional (hysterical) partial ophthalmoplegia.

II. CASE OF INCOMPLETE PARTIAL OPHTHALMOPLEGIA, PROBABLY DUE TO EMBOLISM. REPORTED BY ALFRED WIENER, M. D.

H. S. is 17 years of age. (Fig. II.) Three years ago, while eating his breakfast, he suddenly experienced great difficulty in speaking. This passed away after his meal, and, paying no further attention to it, he went off to school. He suffered all that morning with headache, and, while eating his lunch, again a temporary difficulty in his speech was noticed. This passed off, as the previous attack, without any ill effect. About 4 o'clock the same afternoon he had another attack, and in trying to observe in a looking-glass what the trouble was with his tongue; he

noticed that his face was contracted and pulled toward the left side. This continued for about a minute, and then he dropped to the floor, and became unconscious with an attack of convulsions. After the attack of convulsions he fell asleep; slept for one hour, and awoke apparently well, with the exception of the condition present in his eyes. Previous to this attack he admits having suffered from headache, dizziness and palpitation, which at times were quite severe. Denies all excesses in alcoholism, syphilis and tobacco. Never had rheumatism. Family history negative. Status præsens: Heart is hypertrophied; pulse, 128 to 140; irregular at times. No murmur can be detected, although carefully looked for and examined by several gentlemen besides myself.

The eyes present the following conditions:

Patient's vision is very good. There is a ptosis of the left eyelid. This lid cannot be raised beyond the pupil when the eye is looking straight forward. The right lid is raised only incompletely. The movements of the eye-balls are very much restricted. Diplopia is present when looking either upward or downward, the objects looked at showing one image above the other.

The following muscles are affected in each eye:

Left Eye: External rectus, slight; internal rectus, slight; inferior rectus, slight; superior rectus, marked.

Right Eye: Internal rectus, slight; superior rectus, slight; external rectus, very slight.

The discs and retina in both eyes appear normal. The field of vision and color sense are also normal. Pupils respond to light and accommodation. The patient believes that during all this time the condition above observed has never grown any worse, nor, on the other hand has any improvement been noticed.

The patient otherwise presents no nervous symptoms. He still suffers from headache and dizziness and occasional palpitation.

In regard to the diagnosis in this case, it was necessary at first to determine whether the lesion had only involved

the nuclei of the oculomotor nerve, or actually the nerve itself.

Against the involvement of the nerve, in other words, a peripheral neuritis, was, in the first place, the escape of the intrinsic muscles of both eyes in a bilateral affection; 2nd, a peripheral neuritis is usually unilateral; 3rd, other nerves in close proximity to the third would usually suffer at the same time.

By exclusion we must, therefore, believe that the lesion has involved the nuclei. As arguments against a chronic nuclear ophthalmoplegia, we record the rapid onset, and the absence of any progression in the symptoms showing a further invasion of the nuclei. An acute nuclear palsy of sudden onset is more probable.

In regard to the nature of the lesion, we must differentiate between one of the following conditions: 1st, hemorrhage; 2d, an inflammatory condition which is characteristic of polioencephalitis superior; 3d, embolism.

If a hemorrhage had occurred in this area, the symptoms would have been much more severe and extensive.

In regard to polioencephalitis superior, we are inclined to believe that from the very sudden onset and the absence of any constitutional symptoms, together with the lack of any improvement or progression in the symptoms, that such a morbid process did not exist.

On the other hand, the condition of this boy's heart, together with the sudden onset and the peculiar irregular involvement of the nuclei on both sides, points to the involvement of one of the arterial branches of the basilar artery. The nature of the lesion in an individual of this age, who denies any excess in alcoholism or tobacco, and gives a negative syphilitic history, must necessarily be an embolism.

We have, therefore, in this patient a very unusual and instructive case of an incomplete, bilateral, external ophthalmoplegia of sudden onset, due to an embolus having been lodged in one of the branches of the basilar artery.

A CASE OF MYXŒDEMA TREATED WITH THYROID EXTRACT AND WITH THYROCOLLOID.

Reported from the Clinic of Prof. M. Allen Starr, College of Physicians and Surgeons, New York.

By R. H. CUNNINGHAM, M. D.,

Clinical Assistant in Neurology, Vanderbilt Clinic, Demonstrator of Physiology, Columbia University, New York.

In the British Medical Journal, March 21st, 1896, R. Hutchison, in a preliminary note, claims that the active material of the thyroid gland consists of the colloid matter. In later papers¹ he gives further data regarding the chemistry of the colloid, and also reports two cases of myxœdema that were treated with that substance. Since the publication of his papers I have administered the colloid to a number of thyroidectomized dogs, but it has not been my good fortune until within the past few months to be able to test the activity of that substance upon a patient with myxœdema, or, rather, upon a patient in which the symptoms of myxœdema had very nearly disappeared. As the results are interesting from a therapeutical point of view, and as they apparently corroborate certain facts regarding the toxicity of the thyroid material, which have been described by me elsewhere,² a moderately detailed summary of the history and of the progress of this patient during the treatment may prove of interest to those who have so often remarked the occurrence of toxic symptoms after the ingestion of thyroid extract or of dried thyroid glands.

The patient, J. G., an Englishman from Bolton, æt. 34 years, came under my immediate supervision on February 21st, 1898, in the Neurological Department of Pro-

¹ Hutchison, R.: Brit. Med. Journ., January 23d, 1897; Journ. of Physiol., xx., p. 474, 1896.

² Cunningham, R. H.: Journ. of Exp. Med., iii., 1898.



FIG. I.

fessor Starr, at the Vanderbilt Clinic. From November 22d, 1897, to February 21st, 1898, he had been under the care of Dr. Norrie, clinical assistant in that department, who had treated him very successfully with small doses of the powdered dessicated thyroid of Parke, Davis & Co. Thus, the following summarized history of the patient is from the records of the Vanderbilt Clinic:

November 22d, 1897.—J. G., 34 years of age, brick-layer by trade; chancre in 1886, which was followed by no secondary symptoms, although he received only local treatment for it.

He thinks his present trouble began in 1895, at which time he began to have pains in his back and arms. His arms and legs gradually became weaker, and when he worked he would become fatigued very quickly. In March, 1897, he had to stop work, owing to increasing weakness. He noticed then that his hair was growing thinner. During the past two years his appearance has altered so much that his old friends fail to recognize him. He suffers all the time from the cold, and very often has frontal headache. For one year his gums have been very sore and bleed easily. He weighed formerly about 145 pounds; he weighs now 165 pounds. His appetite is fair and his bowels are regular. His voice has become hoarse and his speech slow.

His hair is thin and dry. The cheeks are flushed, and the skin of the face is firm, that of the arms and legs very firm. The characteristic myxœdematous appearance of the patient is well shown by the accompanying photograph taken by Dr. Norrie. (Fig. 1.)

Temperature of the mouth, 98.5° F.; pulse, 60 beats per minute. Urine contains no albumin nor sugar.

Treatment.—From November 22d to November 26th, he took one five-grain capsule of thyroid (P., D. & Co.) every night. He lost six pounds after taking five capsules.

November 26th —Diarrhœa. Thyroid continued, and a mixture of bismuth, opium and chalk prescribed. De-

cember 1st—No diarrhoea; two capsules per day. December 10th—Weight, 147 pounds. The change for the better in the patient's appearance is marked. Skin moist and perspires freely. December 10th to February 16th, 1898—One capsule per day has been taken during this time. Weight, 143 pounds. Capsules discontinued. February 19th—Weight, 146 pounds. One capsule per day. February 21st—Weight, 145 pounds; pulse, 84; temperature, 98.5° F.; occasional headaches and tightness about the head. He feels almost strong enough to go to work. His hair has returned. His appearance has remarkably altered for the better. (Fig. 2.) Capsules of dried thyroid discontinued and .05 gram of thyroid-colloid (A³) ordered to be taken, t. i. d., between meals.

February 28th—Weight, 146 pounds; pulse, 72; temperature, 95.5° F.; dose of colloid (A) increased to .1 t. i. d. (1 gram of colloid is equivalent to 1 gram of raw thyroid, according to Hutchinson).

March 11th—Weight (shortly after his dinner), 146½ pounds; pulse, 72; temperature, 98.5° F.; ordered .725 gram of colloid (A) t. i. d.

March 25th—Weight, 142 pounds, after discarding his overcoat; pulse, 74; temperature, 98.5° F. Since the 18th has been working hard every day as a bricklayer. He has had no disagreeable symptoms, and has voluntarily remarked that these new capsules don't seem to affect his head, like those he took at first, in November, 1897. Colloid (A) discontinued.

March 28th.—Weight, 142 pounds; pulse, 78; temperature, 98.5° F.; ordered colloid (B³), 1 gram t. i. d.

³ The colloid A was prepared from raw sheeps' thyroid glands that had been removed from the animals about one hour before by the following process: The glands were finely ground and mixed with a large volume of .1 per cent. caustic soda, and the mixture constantly stirred for one hour. The mixture was filtered through muslin and the filtrate precipitated by adding acetic acid. This precipitate, or



FIG. II.

March 31st —Weight, 140 pounds shortly after dinner; pulse, 105; temperature, 98.5° F. He says that during the past two days he has had headache and pains in his back and arms. Colloid (B) discontinued.

April 18th —Weight, 145 pounds; pulse, 74; temperature, 98.5° F. Has been working steadily and feels in excellent health. Ordered .8 gram of colloid (A) t. i. d. (equals 24 grams of raw thyroid per day).

April 25th —Weight, 144 pounds; pulse, 76; temperature, 98.5° F. No disagreeable symptoms have been noticed, and the patient says he feels like a new man. Since the above date I have not seen him, as he is working out of town.

Remarks:—Owing to the fact that the administration of the colloid was begun at a time when, practically, all of the signs of myxœdema had nearly disappeared from the previous use of dried thyroid, its action in the preceding case by no means strikingly demonstrates that it is the active substance of the thyroid gland. It certainly appears to be active enough in preventing a recurrence of the symptoms of myxœdema, and the progressive improvement in the patient's condition seems to continue from its use. A more definite test of its activity would have been, of course, to begin the treatment with the purified colloid; but, judging from its beneficial action in the cases of Hutchinson that were treated at the outset with the colloid, I think it highly probable that, had the above mentioned man been put upon the colloid in the beginning, the result would have been the same.

Besides the very favorable result effected by the treat-

colloid, was collected on filters and redissolved in dilute caustic soda solution. This alkaline solution was then brought to the boiling point, and then reprecipitated by adding a few drops of acetic acid. This precipitate was then collected, spread on glass plates, and quickly dried in a strong current of air, and finally pulverized. After the first extraction a large volume of dilute caustic soda solution was again added to the thyroid material, and the maceration allowed to continue for eighteen hours. From this mixture colloid B was obtained by the method employed to obtain colloid A.

ment, the further very interesting feature in the case consists of the non-appearance of any symptoms of the thyroid intoxication while he was taking comparatively colossal amounts of the colloid (A), the colloid that is removed from the fresh glands before any evident post-mortem chemical alteration of its constituents has occurred. Although it is conceivable that the chemical manifestations, the acidification, etc., which were employed to extract this substance from the fresh raw glands might have produced more or less chemical change in the structure of the colloid, and so have altered its physiological action, no evident change seems to have occurred.

Colloid (A), even in doses equivalent to 24 grams of raw thyroid, did not produce headache, restlessness, tremor, heightened temperature, and acceleration of the pulse rate; or, in other words, the various symptoms of the thyroid intoxication. But comparatively very small doses of colloid (B), which was extracted from the same thyroid material as colloid A during a period of eighteen hours, very promptly sent the pulse rate up to 105, and also produced other symptoms of a mild thyroid intoxication. Practically similar, though more marked, effects were noticed by Hutchinson in his cases of myxoedema to whom very small doses of the eighteen-hour colloid were given. Evidently, then, colloid B contains something that colloid A does not contain, and, as I have previously pointed out,⁴ this something must develop as the result of chemical changes taking place in some one or other constituent of the thyroid during the long period of its maceration. Consequently, the results described in this report fully accord with those that I have described elsewhere, and this observation evidently further substantiates the conclusion reached in my article on experimental thyroidism that the symptoms of induced thyroidism are manifestations of an intoxication resulting from the ingestion of thyroid ma-

⁴ Cunningham, l. c.

terial that has undergone more or less chemical alteration in some one or more of its compounds. Whether or not this change consists of a reversion of some of the less perfectly formed colloid back to the form in which it is taken from the blood, according to one of the early theories of the thyroid function, I am not prepared at present to state definitely, for the chemistry of the thyroid gland, as is evident from the generous profusion of various pure (?) active principles, supposedly existing in the thyroid gland, is still too indefinite for one to venture a decided opinion.

134. PRIMARY SPINAL PERIMENINGITIS. Mollière and Perret (Lyon Medical, May 30th, 1897).

Suppurative inflammation limited to the loose tissue surrounding the dura mater of the spinal cord, is exceedingly rare. Cases thus far reported have nearly all been secondary to caries of the vertebræ, traumatism, bed-sores, accumulations of pus in other parts of the body, or acute infectious diseases.

The case reported by the above authors was that of a young man of 20, who, after being exposed to wet, was taken with pain and rigidity in the back and severe cramps in the lower extremities. He was seen by the reporters on entrance into the hospital three days after the onset, when he had a temperature of 40.4 deg., an elevated pulse, and marked signs of some spinal affection. There was a rigidity of the spine, but no opisthotonos and no spasms; the extremities were not paralyzed, although he could not stand or raise himself in bed. The legs were still the seats of very painful muscular cramps; there was slight exaggeration of the knee-jerks, normal sensation, distension of the abdomen, constipation and retention of urine. Treatment was without effect. The patient's general condition rapidly became worse, and he died 17 days after admission to the hospital, the pain and rigidity of the back resisting to the last. The muscular spasm disappeared, but there was no paraplegia.

At the post-mortem examination the brain and its meninges were found to be normal, and the thoracic and abdominal organs showed only existence of general infection. On incising the back muscles preparatory to removing the spinal cord, pus oozed from the intervertebral spaces, and on removing the arches, the entire cord from its third cervical nerves to the cauda equina was found to be bathed in pus. The dura was somewhat thickened, but its inner surface as well as the spinal pia mater was found to be simply congested, with here and there small traces of pus. The spinal cord itself was absolutely normal, as were also the nerve roots.

PATRICK.

Critical Digest.

ON MULTIPLE SCLEROSIS, WITH ESPECIAL REFERENCE TO ITS CLINICAL SYMPTOMS, ITS ETIOLOGY AND PATHOLOGY.

By B. SACHS, M.D.

(*Concluded.*)

ETIOLOGY.

Opinions have varied much regarding the cause or causes of multiple sclerosis. At the very outset, it will be well to enter a protest against the supposition that the disease, occurring at widely different periods of life, should or could be due to one cause only. But, strangely enough, whenever any data are collected in favor of proving the existence of some special etiological factor, that special factor, in the minds of many, becomes the sole cause of the disease. The study of the subject has led the present writer to the belief that there are several and equally important causes which may give rise to multiple sclerosis. Charcot and his immediate followers recognized the importance of exposure to cold and wet. In recent years little evidence has been brought forward—if we except a contribution by Krafft-Ebing—to substantiate this doctrine; but we may well concede that occasionally it is the refrigeration process, and not a mere wetting, which may be the cause of the disease. The same school attributed multiple sclerosis to traumatism and emotional depression. Mendel has adduced further proof that the disease is at times due to traumatism, basing his views upon four cases of his own and a collection of 24 others taken from litera-

ture. It will be remembered that of late years several authors have also contended for the traumatic origin of tabes. Mendel concedes that there must be an acquired or congenital predisposition to the disease, without which the injury would not have led to the development of this widespread morbid process.

Of the value of emotional origin I have seen several illustrations, one being the case of a young girl of 18, who developed typical multiple sclerosis after the death of a parent, and the other, a boy of 12, who was seriously frightened by a fire occurring near his dwelling, and within a few weeks developed the typical symptoms of the disease.

The earliest writers have recognized that disseminated sclerosis, in contradistinction to paralysis agitans, is a disease of early life, the majority of cases occurring before the age of 30, and some of them before the age of puberty. During the past ten years much has been made of the occurrence of the disease in children; but, as we have seen in the preceding section of this paper, the majority of cases should be looked upon with some reserve, though I am not inclined to deny that the disease does occur, however rare it may be. Women were supposed to be subject to the disease more frequently than men. In 39 cases collected by Berlin, 26 were women and 13 men. Such differences may have been accidental, for later statistics have shown a very different relationship between the sexes. In Nolda's collection of 36 cases, occurring in early life, 16 were boys and 10 girls. In Krafft-Ebing's collection there were 58 males and 42 females. In Redlich's experience the number was almost equally divided between men and women—12 males and 11 females. In Stieglitz's statistics there were 17 males and 17 females, the sex of one case not being mentioned. In the 15 cases seen by myself the ordinary division has been entirely reversed, for of these, 10 were males and only 5 females. Such smaller statistics are subject, however, to accidental

variations, and it would not be fair to infer from them that the disease shows any distinct preference for sex.

Of late years the occurrence of multiple sclerosis after infectious diseases in early life has led to the theory of its infectious origin. As long ago as 1878, Leyden insisted upon the importance of preceding infectious diseases. Other authors referred to its occurrence after typhoid fever (Ebstein, Kahler, Pick, Westphal and others); after cholera (Joffroy); after smallpox (Charcot); but Marie has been the warmest advocate of the theory of the infectious origin of multiple sclerosis, and he contended more particularly that the primary change was to be looked for in disease of the blood vessels. According to Redlich, who has gone over this part of the subject very thoroughly, up to the year of 1897 cases in support of this view were cited by Schoenfeld (after diphtheria), by Massolongo (after influenza), by Rolland (after phlegmonous angina), by Williamson (after rheumatism), by Torti and Angelini (after malaria). Redlich cites a case of polyneuritis, in a boy of 17, coming on after a sore throat. The clinical symptoms in this case gradually passed into those of multiple sclerosis. Henschen refers to a case of a girl of 14, who developed the symptoms of multiple neuritis after diphtheria, death ensuing after three months. At the autopsy he found multiple sclerosis and degeneration in the peripheral nerves. This case appears to us to need careful interpretation, for it is, at least, questionable whether or not multiple sclerosis existed for some time before the diphtheria. The rapid development and early death make the case an entirely exceptional one.²

²While this digest was passing through the press an article by Jürgens has reached the writer. In this communication (*Berliner kl. Wochenschrift*, April 4, 1898) the author reports upon the case of a child, six months old, that had had convulsions at the age of three months; repeated convulsions with paralysis of right arm. Child died in convulsions. Whatever else the case may have been (instrumental delivery, etc.!) it was surely not a case of multiple sclerosis. The brain contained "sclerotic patches," in which protozoa were found similar to those found in patches in the heart. The finding is of interest. A pity it is that it has no bearing upon multiple sclerosis.

Among more recent authors, Oppenheim, Leyden, Goldscheider and Sachs have favored the occasional infectious origin of the disease, but do not feel warranted in supposing this etiological factor to be far more important than many others. Marie's views have been opposed more particularly by Redlich and Krafft-Ebing. In the 15 cases observed by the present writer only 2 came on after infectious diseases, and in both of these so long after such a disease that a direct relation, as of cause and effect, could not be established. It is well to bear in mind, however, that if infectious diseases were the chief cause of multiple sclerosis, the vast majority of cases should come on before the age of 15, and such is not the case.

Oppenheim has directed attention to the large number of cases of multiple sclerosis in which chronic metallic poisoning seemed to play a rather important part, basing his views upon the frequent occurrence of multiple sclerosis in those who work in factories in which toxic chemical substances are used—such as lead, tin, aniline dyes, and the like. It was Oppenheim, too, who referred to the importance of a puerperal condition as a predisposing cause.

It may be asserted with some degree of assurance that syphilis is not an important etiological factor, although Jacobson, Moncorvo and Filatow have cited cases in which hereditary syphilis was present, and some others (Michaelow and Schuster) have referred to acquired syphilis; but Redlich is correct in issuing a caution, which the present writer has also uttered, that multiple sclerosis and multiple cerebro-spinal syphilis have so many symptoms in common that the differential diagnosis is not easily established. Whether or not the disease be distinctly hereditary is still in doubt, although Eichhorst has published two cases in which the disease occurred in mother and child. Its occurrence as a family disease has been noted by Friedreich and Erb (according to Krafft-Ebing

and Redlich), and Totzke has mentioned the occurrence of the disease in two members of the same family. But the question naturally arises, since these cases were not substantiated, whether they may not have been cases of hereditary spastic paralysis.

The latest contribution to this subject has been made by Blumenreich and Jacoby, who report on 29 cases of multiple sclerosis—23 in males and 6 in females—and in these cases the infectious diseases, intoxications and traumatism have been the chief etiological factors. They are of the opinion that the original cause may have preceded the outbreak of the disease for many years, and that the disease may even be of congenital origin. It is somewhat surprising to find that alcoholic and sexual excesses are not cited as occasional etiological factors of some importance; for, in the present writer's experience, two cases seemed to be the result of previous alcoholic excess, and in three now under observation no other factor except prolonged sexual excesses can be found. In these three cases, moreover, the clinical symptoms are so typical that there can be no doubt of the existence of multiple sclerosis.

A little reflection regarding the etiology of multiple sclerosis necessarily leads to the conclusion that many different causes may be at work resulting in the development of the disease, provided there be a predisposition to the disease. I cannot agree with Redlich in refuting Strümpell's theory of the endogenous character of multiple sclerosis, and am inclined to think that for a large number of cases, particularly for those occurring early in life, some slight defect in the original development of the central nervous system must be held responsible for the outbreak of the disease, and that the other etiological factors so frequently referred to are to be regarded as the exciting causes. If we adopt this view, the usual resemblance between the cases of multiple sclerosis developing early in life, and the various forms of hereditary and

family affections observed at that period, can be interpreted more readily.

I have also had occasion to observe several cases in which a distinct neurasthenic condition, due to overwork, preceded the development of the symptoms of multiple sclerosis. According to Eddinger's theory,¹ the development of a sclerosis following upon functional exhaustion does not appear to be at all anomalous. And, finally, I may refer to the fact that in one patient of mine, a music teacher, a functional tremor first appeared in the right hand; this was followed by a distinct intention tremor, and from this stage the symptoms passed into those of a typical paralysis agitans. Altogether, the relation of functional to organic nervous diseases would appear to need further attention, for the same sequence of events has no doubt been observed by others, and it is not to be supposed that the diagnosis of a preceding functional condition was erroneously made. Some of the mooted points regarding the etiology of multiple sclerosis acquire additional interest, if we consider them in connection with a discussion of the morbid anatomy and pathogenesis of the disease.

PATHOLOGICAL ANATOMY AND PATHOGENESIS.

The irregular sclerotic patches (plaques) occurring in different parts of the central nervous system constitute the chief anatomical feature of the disease. These "plaques" represent the terminal condition of a morbid process often lasting through a period of years; but a study of "late stages" has helped us as little toward an understanding of the morbid anatomy of multiple sclerosis, as has been the case in regard to poliomyelitis. The process can be understood only, if studied in its earliest stages. Such studies have been made by Ribbert, Cramer, Bikeles, Taylor and Goldscheider.

With some slight exception, the general agreement has been reached that the blood vessels play an important

¹ This theory appears to have received experimental proof; c.f., Report of the Congress f. innere Med., Wiesbaden, 1898.

part in the initial stages of the disease. Goldscheider describes the conditions as presented by a patch in the cervical portion of the spinal cord. The blood vessels are dilated, and the adventitia is infiltrated with small cells and with considerable detritus. In the vicinity of these blood vessels the nerve fibres are swollen, the sheath and the axis-cylinder participating in the swelling. The medullary sheath suffers more than the axis-cylinder; at least, every cross-section exhibits a number of naked and well-preserved axis-cylinders. Goldscheider supposes that the enlarged nerve fibres crowd against each other, causing the disintegration and absorption of the myelin of some fibres, thus leaving room for other fibres to persist in their enlarged (swollen) condition. The changes thus far described are not unlike those occurring in an ordinary acute myelitis.

Taylor is of the opinion that we are not warranted in connecting the vascular changes directly with the sclerotic process, for the patches are not always related to the diseased vessels, and in many plaques the blood vessels are entirely normal, and other portions of the vascular system show no tendency to disease.

As early as 1863 Rindfleisch maintained the importance of disease of the blood vessels. According to him, each plaque contained in its centre a blood vessel with changed walls, denoting a condition of chronic inflammation. Borst found marked changes in almost every blood vessel (hyaline degeneration, narrowing, thickening of the walls, etc.). He also found a condition of hyperlymphosis and lymphstasis, which he supposed caused a destruction of the medullary sheaths; but his views lack support. Redlich found changes in the larger blood vessels, and even in the capillaries; the neuroglia tissue is easily affected in the immediate vicinity of the blood vessels, leading at times to a perivascular sclerosis. Ribbert found in two blood vessels thrombi consisting of leucocytes. A plugging of blood vessels was also noticed by Redlich; but these

findings appear to us to have no especial significance, except that they prove that the vascular system is diseased in some instances. It is to be inferred, from other reports, that the blood vessels often remain entirely normal. The impartial inference necessarily to be drawn is that, while the blood vessel may be the starting point of the morbid process in some cases, it need not be so in all, and we shall find, as we proceed in this discussion, that the terminal findings are the only ones that are entirely similar in all cases, but that there is a difference of opinion as to the intermediate stages by which this terminal condition is reached. Thus, in Goldscheider's case the nerve fibres were the structures particularly affected; while in Ribbert's case the neuroglia cells were chiefly affected, and there was no swelling of the nerve fibres.

If the nerve fibre becomes involved, it is no doubt true, as was recognized by the earliest investigators (Rindfleisch, Charcot, Leyden, Schultze) that the medullary sheath is easily destroyed, while the axis-cylinder remains normal. Popoff alone contends that the axis-cylinder is also destroyed, but that it is regenerated. This *may* occur at rare intervals; it is much more rational to assume that the axis-cylinder is not destroyed, and the preservation of normal axis-cylinders in a mass, exhibiting any number of disintegrating medullary sheaths, argues in favor of this view. The preservation of the axis-cylinder is responsible for the lack of secondary degeneration in multiple sclerosis. Buss has recorded an ascending degeneration of the columns of Goll, and of the direct cerebellar tract from the eighth cervical segment into the medulla oblongata. These observations are quite unique, and need further corroboration.

It has been assumed by Redlich and others that the fine fibrils which Popoff took to be regenerated axis-cylinders are part of the proliferating neuroglia tissues. It is also significant that Weigert, whose opinion is of the highest value in this matter, insists that the proliferation of neu-

roglia tissue is more marked in multiple sclerosis than in any other disease. This statement militates against the view of Lapinsky, that the fine fibrils discovered in the plaques are connective tissue formations issuing from the medullary sheaths, and are not neuroglia fibres. Recent investigations, utilizing Weigert's neuroglia stain, have satisfied Redlich that the proliferating interstitial tissue consists of neuroglia fibres of varying sizes. Huber concedes that there are slight changes in the neuroglia, but that these consist chiefly of lacunæ, due to the loss of nerve fibres, and of the presence of detritus and granular cells. According to Huber, the process is of parenchymatous origin, the changes in the neuroglia and in the blood vessels being secondary. Adamkiewicz has also advocated the theory of the primary affection of nerve fibres.

If the neuroglia be affected, primarily or secondarily, its cells, as well as its fibres, undergo proliferation. Redlich, who has studied this tissue carefully, maintains that the neuroglia changes may be exhibited in various ways: 1. The increase is largely due to the increase of neuroglia fibres, which gradually form a network of fibres, in which only slightly altered nerve fibres will be found. As these neuroglia fibres grow broader and broader, the network becomes denser. 2. In some patches the network is not so dense, the neuroglia cells and their processes being the most pronounced features, while the nerve fibres have dropped out. This appearance is entirely similar to the account given by Huber, to which we have referred above.

Fuerstner is of the opinion that the neuroglia cells are developed from leucocytes, thus bridging over the chasm between those who maintain that the disease is a parenchymatous affection and those who believe it to be of vascular origin. The actual participation of the neuroglia tissue has acquired greater interest, in view of Struempell's recent theory that the disease is due to a primary proliferation of the neuroglia, that multiple sclerosis is practically a multiple gliosis, and that it belongs

to the order of endogenous affections which are excited by accidental causes, such as injuries and infectious diseases. Ziegler appears to have entertained a similar view (Redlich).

To complete the record of histological changes, we may add that Gowers referred to the involvement of the ganglion cells in the earlier stages of the disease; while Taylor states very positively that the ganglion cells do not become affected until the late stages of the disease have been reached. He draws this inference from the conditions as observed in three cases of his own. The changes as described by Taylor are: Considerable pigmentation, filling up the entire cell body, which is unusual in healthy young individuals; the nuclei are generally wanting; the nerve processes are well preserved; a diminution of cells. Koepfen found the ganglion cells normal. Obersteiner allowed that "the ganglion cells . . . may exhibit changes similar to those found in myelitis." (Quoted after Taylor.)

The patches of disseminated sclerosis present a bluish-gray appearance, and are sometimes slightly elevated; at other times the parts in which they occur appear shrunken and contracted. The question has arisen whether there is any rule governing the distribution of the plaques. By some it was supposed that the brain or the spinal cord may be affected singly. Charcot supported the view of a spinal form of multiple sclerosis. Erb accepted this on clinical grounds only. Charcot, Struempell, Erb and many other writers considered that the white substance of the brain and spinal cord was the favored site; that the gray matter was rarely involved. Charcot believed that the cortex of the brain and cerebellum is rarely affected, and it cannot be denied that in this form the statement is true enough. Basing his conclusions upon three cases, Taylor finds: 1. White and gray are affected without distinction. 2. There is no especially favored site for the development of the sclerotic foci. 3. The cortex of the cerebrum and

of the cerebellum is not exempt. The points 1 and 2 cannot be accepted unconditionally, as such statements must rest upon the examination of a larger number of cases. Charcot believed that the auditory, the olfactory and the optic nerves are the only cranial nerves affected; others (Cruveilhier, Skoda, Taylor) have found all, or almost all, involved. It is well known that the motor and sensory roots of the spinal nerves are often diseased, and Taylor has demonstrated the occurrence of a marked degeneration of the nerve fibres in the cauda equina.

The sclerotic patches *may* occur anywhere, but there can be little doubt that they *do* occur most frequently in the dorsal half of the pons, of the medulla oblongata (whence the occurrence of "bulbar forms"), in the white strands near the periphery of the cord and in the gray matter near the central canal. Obersteiner believes that the patches are lessened in number as we approach the lumbar portion of the cord. Leyden and Goldscheider describe a condition of diffuse sclerosis, or of a diffuse chronic myelitis, due to the confluence of numerous foci in the spinal cord. These authors also describe a purely spinal form of multiple sclerosis, which is the representative of the old-time chronic myelitis.

The sclerotic patches appear, on macroscopical examination, to be sharply differentiated from the surrounding healthy tissue, but, microscopically, the transition is a gradual one. Taylor has shown, however, that the division between healthy and diseased tissue may be strictly defined, and the same is true in Goldscheider's case. (See Fig. 2 of his article.)

The pathogenesis of multiple sclerosis is still obscure. There are theories enough, but none that explains satisfactorily all the cases. In all probability there is no single mode of origin.

Charcot believed that the disease was due to an interstitial inflammation, with a special proliferation of the neuroglia, the destruction of the parenchyma being sec-

ondary to this. Charcot ascribed some importance to the blood vessels, and regarded them as a source of irritation, thus following, in the main, Rindfleisch's teachings. But the part played by the blood vessels did not attain to its present dignity until Marie published his views regarding the infectious origin of disseminated sclerosis, for he supposed that the exciting agents were carried through and along the blood vessels. Ribbert also favored the theory that the plaques were due to some noxious substances conveyed by the blood, and setting up circumscribed inflammatory lesions in different parts of the central nervous system. Mendel explained the traumatic etiology of some cases through the agency of blood vessels. Fuerstner brought the blood vessels into play by making them primarily responsible for nutritive disturbances in the nerve cells and fibres. Goldscheider says: "I believe I shall describe the nature of the morbid process most correctly by assuming the existence of a perivascular inflammation which injures the neighboring nerve structures, causing more especially a disintegration of the medullary sheath, but allowing the axis-cylinder to escape. Later on, in the course of the disease, a reactive interstitial proliferation is established." He regards multiple sclerosis as a form of disseminated myelitis. Koeppen, Huber and Taylor do not attach so much importance to the blood vessels, and Redlich is not convinced that the disease is always of inflammatory origin.

The vascular theory has been opposed by others. Adamkiewicz regards the disease as a parenchymatous degeneration. Weigert is inclined to the belief that the neuroglia changes are secondary to the affections of the parenchyma, and Strümpell, as we have seen, is inclined to regard the disease as being due, primarily, to a peculiar defective (?) development of the neuroglia, which is made evident in later years by especial exciting causes.

We hope soon to be able to shed some further light upon this controversy by researches of our own, but an

impartial review of the results published by other authors leads to the conclusion that there is some truth in several of the views advanced. We believe that the vascular theory holds good, particularly for the cases developed after infectious diseases, after injuries, etc.; and, according to the intensity of the toxic agent, the nerve fibres or the neuroglia may be attacked first. This is in agreement with views expressed by Redlich. Congenital anomalies (Ziegler, Struempell) may make the central nervous system an easier prey for the various toxic agencies and other injuries. But there still remains a considerable number of cases which cannot be explained on the supposition of an inflammatory, toxic or traumatic origin, nor as the result of congenital anomalies. These would seem to depend rather upon functional exhaustion, and such exhaustion might affect primarily the nerve elements of the central nervous system. For such cases the assumption of a parenchymatous affection would be most plausible. In spite of the numerous contributions to this subject, further investigation, impartially conducted, will be beneficial. Above all, there is need of careful inquiries into the earliest histological changes in disseminated sclerosis.

BIBLIOGRAPHY.*

- Adamkiewicz: Die degenerativen Erkrankungen des Rückenmarkes. 1888.
 Ameschof (Forme fruste): Neurol. Centralbl., 1895, p. 732.
 Bass: Das Gesichtsfeld, etc. Stuttgart, 1896.
 Babinski: Etude anatomique, etc. Thèse de Paris, 1895.
 Bechterew: On Specific Disseminated Sclerosis, etc. Archiv f. Psychiatrie, vol. xxviii., 1896.
 Berlin: Deutsches Archiv f. klin. Med., 1874.
 Bikeles: Obersteiner Arbeiten, Vienna, 1895, 3d part.
 Blumenreich & Jacoby: Deutsche med. Wochenschrift, 1897, No. 28.
 Borst: Ziegler's Beiträge zur path. Anat., vol. xxi., 1897 (Hyperlymphosis, etc.).
 Boulogne, P. (de Lille): Revue de Medicine, 1896, p. 404.
 Bourneville et Guérard: De la sclérose en plaques disséminés. Paris, 1896.
 Bristowe: Med. Times and Gazette, 1897.

*For full reference lists consult articles by Redlich and Stieglitz.

- Bruns: *Neurolog. Centralbl.*, 1896, p. 520 (Relation to Myelitis).
 Buzzard: *British Med. Journal*, 1893.
 Buss: *Deutsches Archiv. f. klin. Med.*, vol. xlv., 1889.
 Charcot: *Gazette Médicale*, Jan. 8, 1878. *Progrès Médical*, 1879.
 (Formes frustes.)
 Charcot: *Clinique des Maladies du Système Nerveux*, 1892, p. 399, vol. i.
 Critical Digest: *Brain*, 1896, p. 388.
 Daumtsch: *Neurolog. Centralbl.*, 1890.
 Dejerine: *Revue de Méd.*, 1884.
 Dreschfeld: *Med. Times and Gazette*, 1878, vol. i., p. 140.
 Eichhorst: *Virchow's Archiv*, vol. xcvi., 1896. (Hereditary form.)
 Fromann: *Untersuchungen über die Gefäßveränderungen*, 1878.
 (Quoted by Redlich.)
 Freund: *Archiv f. Psychiatrie*, 1891, vol. xxii., p. 317.
 Fürstner: *Neurolog. Centralbl.*, 1895, p. 615. *Archiv. f. Psychiatrie*, vol. xxx., p. 1.
 Goldscheider: *Zeitschrift f. klin. Med.*, vol. xxx, p. 417.
 Grasset et Rauzier: vol. i., p. 796.
 Gunther Nagel: *Münchener med. Wochenschr.*, 1897, No. 32.
 Gutzler: *Dissertation*, 1895. *Neurol. Centralbl.*, 1896, p. 561.
 (Traumatism as an Etiological Factor of Multiple Sclerosis.)
 Hackney: *British Med. Journal*, March 6, 1897, p. 586.
 Henschen: *Revue Neurologique*, 1897, p. 195. *Centralbl. f. Nervenheilk. u. Psychiatrie*, 1897, p. 298.
 Hoedermaker: *Deutsches Archiv f. klin. Med.*, 1897, vol. xxiii.
 Huber: *Virchow's Archiv*, 1895, vol. xcv.
 Jacobson: *Neurolog. Centralbl.*, 1895, p. 736 (Relation of Syphilis to Disseminated Sclerosis).
 Kahler u. Pick: *Prager Vierteljahrsschrift*, 1897.
 Kaiser: *Diss. Berlin*, 1889. *Neurolog. Centralbl.*, 1889, p. 434
 (Trauma als ätiologisches Moment).
 König: *Berl. kl. Wochenschr.*, 1895, No. 39.
 Köppen: *Archiv f. Psychiatrie*, 1886, vol. xvii.
 Krafft-Ebing: *Wiener klin. Wochenschr.*, 1895, No. 51.
 Kröger: *Neurolog. Centralbl.*, 1889.
 Kunn: *Ueber Augenmuskelstörungen bei der multiplen Sklerose*. Hamburg-Leipzig, 1896.
 Lapinsky: *Zeitschr. f. klin. Med.*, vol. xxviii., 1895.
 Leyden: *Klinik d. Rückenmarks-Krankheiten*, Berlin, 1875, vol. i.
 Leyden-Goldscheider: *Nothnagel's Pathologie*, vol. x., 1897, p. 448.
 Luebbbers: *Archiv f. Psychiatrie*, vol. xxix., p. 768.
 Marie: *Revue de Méd.*, 1883, p. 536.
 Marie: *Maladies de la Moelle*, Paris, 1892, p. 108.
 Massalongo, e. Sylvestri. *Revue Neurologique*, 1893.
 Mendel: *Deutsche med. Wochenschr.*, 1897, No. 7.
 Nolda: *Archiv f. Psychiatrie*, vol. xxiii., 1892 (Relation to Infectious Diseases).
 Obersteiner: *Nervöse Central-Organen*, etc., 3d edition, 1896.
 Oppenheim: *Berl. kl. Wochenschr.*, 1888, 1891, 1896. *Neurolog. Centralbl.*, 1896, p. 43.
 Ormerod: *St. Barth. Hosp. Reports*, 1889, vol. xxiv.
 Popoff: *Neurolog. Centralbl.*, 1894, p. 321.
 Pritchard: *Keating's Cyclopedia of Diseases of Children*, vol. iv.
 Redlich: *Wiener klin. Rundschau*, 1895, No. 49-51.
 Redlich: *Centralbl. f. allgemeine Pathologie u. pathologische Anatomie*, vol. viii., 1897 (containing recent literature).

Redlich: Arbeiten aus Prof. Obersteiner's Laboratorium, 1896, No. 4 (Zur Pathologie der multiplen Sklerose, etc.)

Rendu: *Gaz. Med. de Paris*, 1894, No. 52. *Neurolog. Centralbl.*, 1895, p. 734.

Ribbert: *Virchow's Archiv*, vol. xc.

Rindfleisch: *Virchow's Archiv*, vol. xxvi., 1863.

Rossolimo: *Deutsche Zeitschr. f. Nervenheilk.*, vol. xi., 1897.

Sachs, B.: *N. Y. Med. Journal*, Sept. 1, 1894; *Nervous Diseases of Children*, 1895, p. 345; *Philadelphia Med. Journal*, Feb. 5, 1898 (The Relation of Multiple Sclerosis to Multiple Cerebral Syphilis and to Paralysis Agitans).

Schönfeld: *Diss.* Berlin, 1888 (After Diphtheria).

Schultze: *Neurolog. Centralbl.*, 1884 (Preservation of Axis Cylinders).

Schultze: *Virchow's Archiv*, vol. lxxviii., p. 120 (Relation to Paralysis Agitans).

Schwartz: *Die Bedeutung der Augenstörungen*, etc., Berlin, 1898.

Schuster & Bielchowsky: *Neurolog. Centralbl.*, Dec. 1, 1897.

Stieglitz: *Am. Journal of Med. Sciences*, Feb., 1898 (Contains Literature of Multiple Sclerosis in Childhood).

Strümpell: *Archiv f. Psychiatrie*, vol. x., No. 3.

Strümpell: *Neurolog. Centralbl.*, Nov. 1, 1896 (Multiple Gliosis).

Taylor: *Zeitschrift f. Nervenheilk.*, vol. v., p. 1, 1894.

Totzke: *Multiple Sklerose im Kindesalter*. *Diss.* Berlin, 1893.

Torti and Angelini: *Neurolog. Centralbl.*, 1893, No. 24.

Uhthoff: *Archiv f. Psychiatrie*, vol. xxi., 1891 (Study of Ocular Symptoms).

Unger: *Ueber multiple Sklerose d. Central-Nervensystem im Kindesalters*. Vienna, 1887.

Weigert: *Beiträge zur Kenntniss der normalen menschlichen Neuroglia*. Frankfurt on the Main, 1895.

Werdnig: *Med. Jahrbücher*, 1888. *Neurolog. Centralbl.*, 1889 p. 432.

Westphal: *Archiv f. Psychiatrie*, vol. ix., vol. xiv., 1883.

Wilson: *Br. Med. Journal*, Nov. 25, 1876.

Witzel: *Revue Neurologique*, 1895, p. 313.

Williamson: *Med. Chronicle*, March, 1894 (Vascular Origin).

Society Reports.

NEW YORK NEUROLOGICAL SOCIETY.

Stated Meeting, May 3d, 1898.

Frederick Peterson, M. D., President.

ADDRESS OF THE RETIRING PRESIDENT.

Dr. B. Sachs, in retiring from the presidency of the society, stated that during the past two years the papers and discussions had covered a very wide range, and that greater attention had been paid to psychiatry and to the presentation of clinical cases than formerly.

ADDRESS OF THE PRESIDENT-ELECT-- NEW PATHS IN
PSYCHIATRY.

Dr. Frederick Peterson, on assuming office, delivered an address, entitled "New Paths in Psychiatry." (See page 444.)

SUTURE OF MEDIAN NERVE.

Dr. B. Farquhar Curtis presented a young woman who, last September, received a small punctured wound of the right wrist. Following that there was anæsthesia of the forefinger and, to a less extent, of the thumb and middle finger, but without any marked paralysis. The area of anæsthesia was not quite typical of that observed after complete division of the median nerve, because the ulnar side of the middle finger was not involved. The operation had been done a week ago, and, on exposing the nerve, it had been found that it had been completely divided, in spite of the atypical area of anæsthesia. On cutting off the bulbous ends an interval of about one inch was left between the ends of the nerves. Both ends were forcibly stretched, and apposed, and the hand kept in a flexed position by a suitable splint. Two or three catgut sutures were passed, not merely through the sheath, but through

the nerve itself. Instead of cutting away the bulbous ends entirely, the fibrous part of the sheath was preserved and used as a sort of flap for the reinforcement of the suture. The anæsthesia had been so complete before the operation that she had repeatedly burned the ends of the fingers.

TRAUMATIC ULNAR NEURITIS—TRANSPLANTATION OF THE NERVE.

Dr. Curtis also presented a woman, twenty-eight years of age, who had been doing a good deal of writing with a pen. In childhood she had received a fracture of both condyles of the right elbow. As a result of this, the ulnar nerve lay upon an exposed surface, instead of in the groove. For two or three years there had been increasing numbness and pain. Dr. Dana had seen the case, and made a diagnosis of neuritis. Dr. Curtis exposed the nerve by a curved incision and flap. The nerve was found to be thickened for a distance of about an inch and a half, and to about twice its normal diameter. On incision through the sheath there seemed to be a general increase of the fibrous tissue of the nerve. The nerve was split and stretched gently, and then removed from its exposed situation to a more protected part, i. e., to the front of the elbow. The result has been surprisingly satisfactory, so that she has been enabled to resume her long hours of writing without discomfort. The speaker said that there had been some cases reported of dislocation of the ulnar nerve, in which the nerve had been restored to its proper groove by operation. There had been no material change in the general appearance of the hand, except perhaps that it was a little more plump.

DISCUSSION.

Dr. Sachs said, regarding the maintenance of sensation in the ulnar half of the hand in the first case, that the areas of sensation pertaining to the two nerves are so irregular that one can not predict with any certainty as to the result in any given case. The result in both cases presented was certainly very good.

TREPHINING FOR EPILEPSY.

Dr. B. F. Curtis then exhibited a recent case of trephining for epilepsy, with a view to illustrating the technique of the general operation of craniotomy with a bone flap. The patient, an Italian, twenty-five years of

age, had had epilepsy quite severely, but the details of the previous history were not in his possession at present. There were no localizing symptoms, but there was a scar on the right side of the head, and, at Dr. Hammond's suggestion, an exploratory operation was performed last March. As there was no definite localization, a large opening was required. This was made by the osteoplastic method, using the wire saw. Four small trephine openings were rapidly made, and then a flat director of German silver was passed between the skull and the dura from one opening to the other. Through this an eyed-probe armed with a string could be passed. The string served to carry the wire saw. The latter is nothing but a piece of piano-wire having a screw thread cut upon it. It is operated like a chain saw, and cuts very rapidly. The advantage over the electric saw was that it eliminated the uncertainties connected with the use of a battery. The disadvantage of having to put a guard underneath was one which is shared by all the other methods of this nature. As the saws were very cheap, a new one could be used for each case. The saw was used on the three sides, and the remainder of the flap broken away. Fully one third of the brain was exposed in this way. It seemed to him much easier to make these large openings and work through them than through the smaller trephine apertures. The cut with the saw is made obliquely, so that immediately afterward the bone can be replaced and will rest firmly in place. In this case some atrophy of the cortex and of the underlying parts was found.

Dr. Curtis then reported two other cases which had been operated upon previously, and which had failed to present themselves at this meeting for inspection. The first case was that of a married woman, thirty years of age, who, as a result of a fall, had severely injured her right arm. She did not have any epileptic fits, however, until six years before coming under Dr. Dana's observation. Shortly before that time they had become very frequent and severe. In other respects she appeared to be healthy. The attacks always began in the left foot and spread up the leg; then they attacked the arm and spread to the body. She frequently had a complete, general and typical epileptic attack, with loss of consciousness. There was no paralysis and no optic neuritis. She would have as many as a dozen attacks in one night. The speaker said that at

the operation he had made an opening, two inches in diameter, over the fissure of Rolando. On testing the centres with a strong current of electricity an intense general convulsion had been provoked. This began in the left leg, and the pulse becoming very feeble, the wound was closed temporarily. It was worthy of note that the fits continued, notwithstanding the relief of pressure afforded by the removal of the bone. A week after this operation the wound was reopened, and a flap of dura was turned back. The vessels surrounding the motor centres were ligated, and then the cortex on both sides of the fissure of Rolando and the region of the arm and leg centres was excised. Some adhesions to the pia mater were encountered and separated. There was some rise of temperature, but no other sign of inflammation after the operation. The patient was completely paralyzed in the left arm and leg, and had a few fits in the first week, and her whole demeanor underwent a marked change. After a time the mental depression passed off, and when she left the hospital she was in good spirits. She took the bromides during her stay in the hospital, but discontinued them afterward. Motion in the arm and leg began six weeks after operation, and in six months she had complete power in both extremities. Eighteen months after operation he had examined her. She had gained twenty or thirty pounds in weight, and had entirely recovered the use of the hand and leg. She no longer had fits, and was in the best of health, her former cheerfulness and mental activity having been restored. Slight headache and indisposition to meet people at certain times were the only remnants of the old trouble. There was a considerable depression of the scalp over the aperture.

The second case was one of hemorrhagic cyst of the brain, with epilepsy, in which recovery followed the operation of trephining. The patient was a Russian boy, thirteen years of age, who was admitted to the Post-Graduate Hospital on June 11th, 1896. Fourteen months previously he had had a cerebral hemorrhage, with left hemiplegia, and for about a year he had suffered from epileptic fits. There was no special localization, but he was guided to the situation by the limited paralysis on the opposite side of the body. In this case the operation was about the same as in the other case. The aperture was made about two inches in diameter over the motor region. The dura

was opened, and when the circulation became feeble, the brain appeared unusually blanched and soft. Further exploration revealed a cystic cavity immediately underneath the cortex. Here the cortical substance of the brain was very much thickened by fibrous tissue. The cavity extended backward into the posterior lobe, and forward for three or four inches. The cyst contained clear serum, but there was no tension in the cyst. The cyst was closed by catgut sutures, as the brain tissue was quite fibrous and permitted such a method to be used. The operation was done on July 4th. On October 25th there were seven convulsions in twenty-four hours. On the following day a large aspirating needle was plunged into the cyst, and five or six grammes removed. He remained free from convulsions until February 18th, 1897, when it became necessary to repeat the aspiration. Up to last January he had remained free from convulsions under the use of the bromides.

DISCUSSION.

Dr. W. M. Leszynsky said that he had seen this boy about one week ago. Although before operation the bromides had had absolutely no effect, since the last aspiration of the cyst the boy had been comparatively well. He had been taking thirty grains of bromide, three times a day, and now had only an occasional slight attack, limited to the paralyzed side—about one in three weeks—and his mental condition had improved. He had reported the case to the American Neurological Association last year. Even in such apparently hopeless cases some benefit seemed to follow surgical interference. He had suggested an exploratory operation, in view of the limited character of the paralysis.

Dr. C. L. Dana said he desired to emphasize the remarkable success achieved in the case which had been under his observation, and which was one of true, classical Jacksonian epilepsy, without any discoverable gross lesion of the brain. Dr. Collins and he had made a microscopical examination of the excised part, and had found marked degenerative changes in the cells. Dr. Collins had published in "Brain" the results of this study. Dr. Dana said he had no doubt that the case, if left alone, would have developed into one of ordinary epilepsy.

Dr. Sachs said that one great obstacle to the successful performance of these operations had always been the difficulty of obtaining a suitable saw. The instrument just exhibited seemed to be a great improvement on former instruments. Most of the neurologists and surgeons seemed now agreed upon the advisability of using a large flap. He thought a care-

ful record should be made of every case of this kind in which a good result had been obtained, because the whole subject of the surgical treatment of epilepsy had lately received another serious blow from Bergmann. Certainly, a sufficient number of cases had been relieved by operation to warrant a selection of such cases as seemed suitable for operation, particularly those in which the interval between the development of the epilepsy and the operation was not very long. The fact that in the case just reported the interval had been six years made the good result all the more remarkable.

Dr. J. Arthur Booth said that it was common for cases to go without attacks for two or three years under the bromide treatment, and without surgical interference; hence, these cases should not be considered as cured until at least four or five years had elapsed.

Dr. Peterson also thought it extremely important that careful records of these cases should be kept for a long time after operation. At least four years should elapse without an attack before a case should be considered as cured.

Dr. Curtis said he had not intended to bring up the general subject of the surgical treatment of epilepsy at this time. He thought the surgeon should be willing to operate upon a good many of these cases, but two classes should be carefully distinguished, viz., (1) cases like the ones just reported, in which there is localization of the epilepsy, and (2) cases like that of the Italian just presented, whose future is hopeless, and whose history is vague. For the sake of the few who might be benefited it seemed to him justifiable to operate. The operation certainly causes a temporary amelioration of the symptoms, and this repays the patient for the slight risk incurred by submitting to the operation. In cases requiring an extensive and dangerous operation the probable benefit is correspondingly great. He was not enthusiastic about this kind of work, and did not speak of the cases reported as "cured," but it was certainly worth while to place them on record. At present an exploratory operation seemed to him justifiable and desirable.

PHILADELPHIA NEUROLOGICAL SOCIETY.

March 28th, 1898.

The President, Dr. F. X. Dercum, in the chair.

Dr. James J. Putnam, of Harvard University, read a paper on

THE NATURE AND SYMPTOMS OF THE POST-TRAUMATIC NEUROSES, BASED ON PERSONAL OBSERVATIONS. (To be published in this journal.)

DISCUSSION.

Dr. Charles K. Mills said that the society was certainly under great obligations to Dr. Putnam for the manner in which this subject had been presented. He had brought before us, in a manner somewhat different from that in which the subject is usually treated, the importance of the psychical element in the production of the condition present in post-traumatic neuroses. All who have had much experience with these cases appreciate the importance of this element.

The points that he had made with reference to the social condition of the sufferers from these injuries were novel and of interest. They had not been brought forward in the same way in the discussion of these cases, although Dr. Putnam may, perhaps, have laid almost too much stress, relatively, upon the psychical or moral element.

Dr. John K. Mitchell was glad to be able to reinforce from a recent observation the statement of Dr. Putnam as to the frequency with which these cases are seen in certain classes. The difficulty of treatment is also increased by this very element of want of energy. As has been said, traumatic neuroses are more apt to occur, and are more apt to be unsatisfactory in treatment and result, in what we may refer to as the lower middle and lower classes. He had a patient of this kind who had given him a great deal of trouble, and, while her physical condition had become normal, she had no reserve of energy to enable her to recover from the neurosis. He thought that this want of energy is partly a congenital condition. Attention had not been before so forcibly called to this matter, but it is of interest both as regards causation and treatment.

Dr. F. X. Dercum said that his experience differed in es-

sential points from that of Dr. Putnam. In the first place, by far the larger number of cases present evidences, marked or slight, of some physical injury—most frequently to the muscular and fibrous structures (ligaments, muscular insertions, fasciæ) of the back, neck, other portions of the trunk, limbs, etc.; furthermore, cases in which the traumatic neuroses are associated with gross physical injury to the skull, bones or joints are not by any means infrequent. Secondly, when nervous symptoms are present in these cases they always assume a definite and well-defined syndrome, either that of neurasthenia or that of hysteria; sometimes the stigmata of both conditions are present. Thirdly, in Dr. Dercum's experience, the statement made by Dr. Putnam with regard to the social condition of persons presenting the traumatic neuroses is not borne out. Traumatic neurasthenia and traumatic hysteria occur just as frequently, if not more frequently, in the upper social classes as in the lower. It is merely that the poor and less well-to-do seek redress by litigation more frequently than the rich. The illustration used by Dr. Putnam of the comparative immunity enjoyed by football players is not valid. First, because football players are a trained body of picked athletes, and, most important of all, because the injuries from which they suffer are not accompanied by the element of shock, especially psychic shock.

Dr. Spiller hoped that Dr. Putnam would not think he did not appreciate the value of the paper if he took, to some extent, the opposite side. He thought that there is considerable danger of considering *certain* organic diseases as functional, especially as the number of cases of true traumatic neuroses is so large. He spoke of a case which he had recently seen, in which the diagnosis of a spinal cord lesion could be excluded almost with certainty. A man had received a very severe blow in the lumbar region, and afterward was very weak in the lower extremities, especially in one limb. One might have said that concussion of the spine existed in this case, but this could be excluded after careful examination. No vesical or rectal disturbances, no diminution of the reflexes and no alteration of sensation, even in the perineal region, were noted. A large area of ecchymosis and an abscess were found in the lumbar region. With proper treatment the man was cured within a few weeks. Another patient had been seen with Dr. C. M. Edwards. He had received a severe blow on the head, and had had at first the signs of concussion of the brain. After some weeks he had entirely recovered, except that he had lost the senses of smell and taste. It is not impossible that these symptoms were the result of contre-coup. We have reason to believe that the centres for smell are situated at the base of the cerebrum, and it may be

that those for taste are near the same region, although we know little positively in regard to the latter.

Observations on the lower animals show that organic changes do occur after concussion of the spine without fracture (Schmaus, Bickeles, Kirchgässer), and a number of such cases occurring in man has been collected by Wagner (*Beiträge zur klin. Chir.*, vol. xvi.)

Dr. Spiller said that Dr. Willard and he had reported a case in which fracture of the spinal column had occurred. They had stated very distinctly that the fracture could not be ignored, and they had not attempted to separate the lesions resulting from such a condition from those due to the concussion. They had desired to call attention to the fact that where vertebral fracture exists, some of the symptoms are due to concussion. This view seemed to them tenable, inasmuch as very intense lesions, even rupture of the spinal membranes and cord, have been observed in cases in which no fracture had existed.

Reference was made to the extraordinary paper by Erb, in which poliomyelitis seemed to be the result of trauma. We are beginning to realize that trauma may play a very important part in the development of organic nervous diseases.

Dr. J. J. Putnam stated that in preparing this paper almost all the points that the gentlemen had brought forward were in his mind, but he did not have time to discuss them. He certainly agreed with Dr. Dercum, that persons of the cultivated classes do have these conditions. They suffer from the shock in the beginning, and, it may be, pretty severely, but differences are seen later. The tendency of the disease among them is more often toward the neurasthenic than toward the hysterical type, and there is a greater tendency to improvement, on the whole, at least among the lighter cases.

With regard to what Dr. Spiller had said, he fully agreed that these accidents are sometimes productive of actual lesions. Possibly, slight lesions are of very frequent occurrence, but he did not think that they play an active part in causing the symptoms of traumatic hysterias and neurasthenias. He had a series of cases, which he did not have time to bring up, in which chronic spinal symptoms had followed these injuries. One was that of a lady, 28 years of age, who was thrown from a wagon, striking her head and face. The arms and legs felt powerless for a short time, and then, substantially but not fully, recovered. He saw her fifteen weeks later. The movements of the feet and legs were impaired, and there was slight impairment of coördination of both legs and arms, and some impairment of sensibility, yet there was no fracture nor even stiffness of the vertebral column. He had seen several well-marked cases of this class which corresponded with those reported by Erb.

In presenting a subject like this, one has to emphasize the points that he wishes to enforce, and his object was to bring out strongly the fact that certain psychical factors do play a more important part than has generally been ascribed to them, and to suggest the lines on which this subject should be studied.

Dr. H. A. Hare exhibited

A CASE OF UNIVERSAL MUSCULAR ATROPHY.

(See page 450.)

Dr. Dercum presented

A CASE OF SYRINGOMYELIA OF THE LUMBAR CORD.

As is well known, syringomyelia affects, in by far the greater number of cases, especially the cervical portion of the cord. In the patient exhibited by Dr. Dercum the lumbar portion of the cord was evidently involved, while the cervical portion was apparently free. The case was also interesting because of the presence of trophic ulcerations on one of the toes, and in this respect it resembled another case of syringomyelia of the lumbar cord presented by him to the society a year or two ago. The case is as follows:

The patient was a male, aged 26 years, a clerk, and a native of Roumania. He had had the ordinary diseases of childhood, which presented nothing peculiar. Some ten or twelve years ago he had suffered severely from conjunctivitis. He had also had several severe attacks of supraorbital neuralgia. He had no illness of moment until two years ago, when he noticed a burning on the anterior surface of the left thigh, and several weeks afterward a sensation of heaviness in the left knee. For some time subsequently he experienced occasional shooting pains, extending from the left knee to the foot, and during these paroxysms the foot would jerk. About a year later similar symptoms made their appearance in the right leg, save that no sensation of burning was felt here.

These symptoms had persisted with increasing severity, and had continued to be most pronounced in the left leg. He had suffered also greatly from constipation and indigestion. Three months ago he noticed loss of sensation

in the left leg, below the upper third of the thigh. No history of syphilis was obtained.

The station of the patient at the time of his presentation was somewhat ataxic. The gait was both ataxic and spastic. The left leg was more spastic than the right. The knee-jerks were plus, and ankle-clonus was present. Complete loss of the temperature sense and of the pain sense was found in the left thigh, left leg and left foot. Tactile sense, on the other hand, was everywhere well preserved. No thermal loss or analgesia existed in the right leg or elsewhere. The patient, in addition, complained of soreness and pain, which he referred to the lower portion of the back, and which he stated extended down the hips and thighs. The right knee seemed to be distinctly larger than the left, and suggested, possibly, a beginning arthropathy. On the middle toe of the left foot an ulceration was seen at the base of the nail. This ulceration was perfectly painless. The patient stated that it seemed to be in process of healing, as the sore was smaller than it had been.

135. IL BAGNO D'ARIA CALDO COME MEZZO TERAPEUTICO D'ALCUNI PAROSSISMI EPILETTICI (The Hot Air Bath as a Means of Treating Epilepsy). C. Cabitto (Revista Sperimentale di Freniatria, 23, 1897, p. 52).

In a previous article on the toxicity of the sweat of epileptics, the author showed that the sweat of epileptics during the paroxysm exerted a toxic and convulsive effect when injected into animals, especially rabbits. He, therefore, concluded that the retention of the sweat tended to confirm the theory of auto-intoxication in the genesis of epileptic seizures. The author reports four cases in full, where the hot-air bath was successfully used, and states that in other cases he has had equally good results. The length of time that the patient remains in the bath is about one half-hour. The frequency of the bath depends upon the frequency of the convulsions.

The author concludes as follows:

"The hot-air bath has proven, in my cases, an excellent means to prevent and interrupt the epileptic seizures. There is hope, when other observations come to confirm these results, that such a method, united with lavage and antiseptic treatment of the digestive tract, not excluding other means for stimulating the secretions, will in many cases of general epilepsy prove a logical and useful method of treatment, or, at least, a less objectionable one than the bromide treatment.

KRAUSS.

Periscope.

With the Assistance of the Following Collaborators:

CHAS. LEWIS ALLEN, M.D., Wash., D.C. R. K. MACALESTER, M.D., N.Y.
J. S. CHRISTISON, M.D., Chicago, Ill. J. K. MITCHELL, M.D., Phila., Pa.
A. FREEMAN, M.D., New York. H. PATRICK, M.D., Chicago, Ill.
S. E. JELLIFFE, M.D., New York. JOSEPH SAILER, M.D., Phila., Pa.
WM. C. KRAUSS, M.D., Buffalo, N.Y. HENRY L. SHIVELY, M.D., N. Y.
W. M. LESZYNSKY, M.D., New York. A. STERNE, M.D., Indianapolis.

ANATOMY.

136. ON THE STRUCTURE OF THE NEUROGLIA. James R. Whitwell
(British Med. Jour., i., 1898, p. 681).

This is a beautifully concise statement of the present status of the neuroglia question, with a few suggestions based on a new method of examination. This method is to swell the section of nervous tissue by brief immersion in a hot concentrated solution of caustic potash and then to allow it to dry, which optically obliterates cells and their processes and brings into clear relief the neuroglial mesh-work. The method attains, indeed, the same object as a differential stain, such as that of Weigert; that is, it brings out strongly certain tissue elements to the exclusion of others. The author does not state that such a method has its draw-backs as well as advantages. In the present instance the former are serious, for with cells and cell processes excluded from vision, the question of the connection of neuroglia fibres with glia cells, could scarcely be determined. His investigations, however, are valuable especially as he has employed his method in connection with the Golgi stain. He concludes:—

1. The sustentacular apparatus of the nervous system consists of an interlacing net work of fibrils, in the meshes of which lie cells both neuroglial and nervous.
2. These fibrils show no evidence of being direct processes of cells, and do not appear to branch.
3. The sponge-like reticulum thus formed is to be regarded as the peripheral portion of the lymphatic system, consisting of lymphatic spaces and channels.
4. The fibrils form a complete basket-work for each element in the nervous tissue, including the blood vessels.
5. The fibrils consist of a highly refractile substance (refractive index about 1.5) with a considerable degree of elasticity, as is shown by their curvature on release.
6. Chemically the fibrils appear to be composed of a substance which is neither neurokeratin nor elastin. PATRICK.

137. STUDIES ON THE NEUROGLIA. F. W. Eurich (Brain, 20, 1897, p. 114).

The author, in a short note, discusses the neuroglia tissue from a comparative point of view, in the light of the newer Weigert discoveries. He shows briefly some of the stages in the development

of this tissue through the lower forms and discusses some of the theories of its functions. He believes that Bevan Lewis' "scavenger cell" is but a form characterising the neuroglia cell in one period of its life history, and that in any proliferating process this earlier stage must be returned to, before fibrillation, as the final result can be attained.

VOGEL.

138. A CONTRIBUTION TO THE STUDY OF HUMAN NEUROGLIA. W. Taylor (Jour. of Exp. Med., 2, 1897, p. 611).

On a basis of two cases of cerebral tumor the author, using Mallorey's modification for staining the neuroglia, comes to the opinion that the term connective tissue should be dropped when discussing sclerosis of the nervous system, and that the term "glio-sarcoma" should also be relegated to oblivion. Neuroglia cells show a successive type of differentiation, first possessing no processes, later with processes, and finally these processes developing into true fibrils; thus he adopts Weigert's view. In the tumors under discussion both the latter stages were evident. In contradistinction to Weigert's statements, the author is not prepared to accept that a glioma is characterized by an increase of glia cells, while a gliosis consists of an increase of fibres. He further adds that no distinctive features separate gliomata and sarcomata.

JELLIFFE.

139. NOTES ON GRANULES. A. Hill (Brain, 20, 1897, p. 125).

In using his chrome silver method, the author believes he has found a new type of cell in the cerebellum. These he first described as "granules with centripetal axis-cylinder processes." They resemble Golgi cells in an embryonic condition, but retaining their shape in the adult, he believes them to be a type distinct from the Golgi cells of the cerebellum. These cells were found near the summit of the folia and only in the deeper stratum of the granular layer. In diameter they range from 10-15 mikra. (in the rat), about the same as the ordinary cerebellar granule cells. Their axis cylinders run parallel with the fibres of the arbor vitae, often traversing the granular layer for some distance before joining the fibres. These are usually destitute of collaterals. Most of the cells are carrot-shaped, semi-fusiform. The centripetal axis cylinder process distinguishes them from the granules. The author has found these cells in the cat and in the rat.

JELLIFFE.

140. STRUCTURE OF THE SPINAL GANGLIA OF MAMMALS W. Flemming (Arch. f. Psychiatrie, 29, 1897, p. 969).

Flemming confirms in the main the observations made by Lenhossék upon the structure of the cells of the spinal ganglia. By his method of progressive staining with hematoxylin he maintains, in opposition to Lenhossék, the fibrillary structure of the achromatic substances of the cells. These fibrillated structures are more or less longitudinally arranged at the sites of the origin of the dendrites and, in the main body of the cell, are more reticulated. It is, he believes, characteristic that this reticulum is more marked in the cells of the spinal ganglia than in the cells of the anterior horns or of the cortex.

JELLIFFE.

141. DER ZELLENBAU DER GROSSHIRNRINDE DES AFFEN (MACACUS CYNOMOLGUS) The Cells of the Cortex of the Monkey (Macacus Cynomolgus). M. Schlapp (Arch. f. Psychiatrie, 30, 1898, p. 583).

The author here presents a careful and valuable contribution to our knowledge of the comparative cytology of the vertebrate brain. The investigation is made by the Nissl methods and this investigator's classification of the cell types is followed by the author: In the brain

of the monkey it would appear that there are three fairly well differentiated areas, each with its distinctive grouping of cell elements. The region occupied by the first type comprises the frontal convolutions and is bounded behind by the precentral convolutions. Its cell distribution resembles in some particulars that of the motor region type of man consisting of five layers:

1. The layer of tangential fibres with irregular cell groupings.
2. Layer of the small polymorphous cells.
3. Layer of small pyramidal cells, with small oval and round cells between, most of the cells being found in the para pygnomorph condition.

4. Layer of large and giant pyramidal cells which are usually in the pygnomorph condition.

5. Layer of polymorphous or spindle cells.

The area of the second type occupies the region just posterior to that of the first type, and constitutes the greater part of the cortex, leaving but a small area, homologous with the general occipital region for the third type.

This area is characterized by a seven-layered arrangement of cells. The first and second layers are similar to those found in the area of the first type. The third layer is also, in the main, similar; added to it are a number of cells for which B. Lewis has introduced the name "globose cells."

The fourth layer is the same as the outer half of the corresponding layer for the first type, while the inner half is made up of new type cells, constituting the fifth layer. These cells are the "granule cells"—corresponding to the 4th layer of Hammarberg's sensory type. The sixth layer contains pygnomorph pyramidal cells, similar to those found in the inner parts of the fourth layer of the first type, and the seventh layer is composed of the polymorphous cells.

The area of the third layer is the smallest, yet, perhaps, the most characteristic. It occupies the general region of the occipital lobes, and its cells appear to be arranged in eight more or less regular layers. These, briefly stated, are:

- 1st, 2d, and 3d layers as in the first type.

- 4th layer, reproduction cells, in "granule" cells.

- 5th layer, projection cells, apygnomorph pyramid cells.

- 6th layer, "granule" cells.

- 7th layer, projection cells.

- 8th layer, polymorphous cells.

The paper is well illustrated; and the character, and sizes, and variations of the various cells carefully elucidated. JELLIFFE.

PHYSIOLOGY.

142. A STUDY OF THE TEMPERATURE SENSE. J. F. Crawford (Psychological Rev., 5, 1898, p. 63).

In a preliminary report of experiments upon six subjects, the author comes to a conclusion the opposite of Goldscheider's, as he finds continuous sensitive regions, and within a fairly sensitive region he could not find a spot that was non-sensitive. These sensitive regions are of various size and indefinite limits, and within them are often found smaller regions of greater intensity, but never in the form of groups of spots.

Three of his subjects gave thirty sittings, and three gave eight sittings. He used two pairs of brass cylinders, one pair being $\frac{1}{2}$ mm. across at the ends, and the other pair being considerably less. Sharper points produce the sensation of pricking or burning. The cylinders were partly covered with cork and immersed in hot and cold water of known temperatures.

Slight changes of temperature are insignificant, especially with cold, a difference of 5 deg., making no practical difference in the reactions. From 0 deg. up to 23 deg. or 24 deg. there are cold reactions, intense below 6 deg. or 8 deg., and growing less marked up to 15 deg. At 22 deg. hot reactions begin to come in but are not marked until 40 deg. or 42 deg. are reached. At between 49 deg. and 54 deg. the heat passes over into pain which arises from all points of the skin alike. The range of cold reactions is much greater than that of hot.

The relation between "hot" and "cold" seems to be one of mutual independence. They are neither coincident nor complementary, but seem to overlap without law. The personal factor was prominent.

CHRISTISON.

143. LA TOSSICITÀ DEL SUDORE NEGLI EPILETTICI (The Toxicity of the Sweat of Epileptics). Cabitto (Revista Sperimentale di Freniatria, 23, 1897, p. 36).

Dr. Clemente Cabitto experimented with the perspiration of epileptics upon rabbits and came to the following conclusions:

I. The sweat of epileptics in the prodromal period of the attack injected into the circulation of rabbits provoked a decisive toxic action and a very strong convulsive attack.

II. The toxic and convulsive power of the sweat increases as the attack advances, and decreases in the period after the paroxysm relatively with the duration of the post epileptic state.

III. The attacks preceding the day of experimentation do not exert any influence over the toxicity of the sweat.

IV. The action of the sweat of epileptics some time before the attack does not differ from that of a healthy subject. KRAUSS.

144. LE SENS DE L'ORIENTATION. (The Sense of Position). P. Bonnier (Revue Scientifique, 1898, p. 108).

In a short and interesting article the author discusses the general problem of the sense of position. To the ampullæ of the semi-circular canals he attaches much importance, in that there is a memory sense connected with the displacement of their contents. VOGEL.

145. LA SENSIBILITÉ MUSCULAIRE DES YEUX (Muscular Sensibility of the Eyes). B. Bourdon (Revue Philosophique, 22, 1897, No. 10).

In the estimation of space this muscular sense is of value, and the results of previous investigations having given such contradictory results, the author was led to perform some experiments.

The results of these experiments would be contrary to the hypothesis that our conception of space is due to muscular sensibility of the oculomotorius. The movements of the eyes are, therefore, not controlled by muscular sensibility, but by the retinal impressions. Muscular sensibility of the oculomotorius could be markedly diminished, yet visual space perception suffer no loss. JELLIFFE.

PATHOLOGY.

146. DEGENERATIONEN DER VORDERHORNZELLEN DES RÜCKENMARKS BEI DEMENTIA PARALYTICA (Degenerations of the Cells of the Anterior Horns of the Spinal Cord in Dementia Paralytica). H. Berger (Monatsschrift f. Psychiatrie u. Neurologie, 3, 1898, p. 1).

Berger has examined the cells of the anterior horns of the cord in twelve cases of dementia paralytica. He has found pigmentary degeneration, karyolysis, destruction of the dendrites, tumefaction of the cells, chromatolysis, vacuolation, changes in the nucleus and

nucleolus, etc. The division of the nucleus of the nerve cell into two parts, reported by him, each with a nucleolus, without division of the cell body, is worthy of special note. Berger regards this as an incomplete regeneration of the nerve cell. He has observed changes in both horns of the cord in man after destruction of one pyramidal tract, but he was unable to find that lesions of this tract in the dog and cat, or of a posterior root in the dog, had any effect on the anterior cells of the cord. He explains the muscular atrophy seen in dementia paralytica by the cellular changes which he has observed, and states that such cellular alteration occurs in the anterior horns of the cord in 83 per cent. of all cases of general paralysis, and is most frequent in the lumbar region. He believes that the disease of the anterior cells is primary; that it is, to a certain extent, independent of degeneration of the white columns; and that there is no constant relation between the degree of these cellular changes in the cord and the degeneration in the brain.

SPILLER.

147. THE MORBID ANATOMY OF A CASE OF HEREDITARY ATAXIA. A. Meyer (Brain, 20, 1897, p. 276).

The author had an opportunity to study a case already reported by Dr. Sanger Brown. The following general statements are offered:

1. There is no circumscribed cerebellar lesion, nor does the cortex show a marked decrease of the number of the Purkinje cells.
2. Parts of the spinal cord and medulla which are known to have relations with the cerebellum were found affected.
3. The spinal cord, as a whole, shows increase of the superficial neuroglia, and a remarkably large number of corpora amylacea, similar to what is seen in very old people, in paralysis agitans, etc.

JELLIFFE.

148. FAIT RELATIF A L'ÉTUDE DE LA PATHOGÉNIE DES ARTHROPATHIES ET DES FRACTURES SPONTANÉES CHEZ LES TABÉTIQUES (The Pathogeny of Arthropathies and Spontaneous Fractures in Tabetics). A. Pitres et G. Carrière (Archives Cliniques de Bordeaux, 5, 1896, p. 483).

The authors give the clinical history and pathological findings in a case of tabes in a man of 58, in whom the disease had existed since his twenty-sixth year. At 38, there was spontaneous fracture of the right tenth rib; at 42, arthropathy of the left knee. The autopsy and microscopical examination showed the usual sclerosis of the posterior columns, no change in the antero-lateral columns or the cells of the anterior horns; advanced atrophy of the posterior roots, the anterior roots normal; diffuse alterations in the ulnar, intercostal and nerves of the lower extremities, especially marked in the nerves of the left knee-joint, and the tenth right intercostal—supplying the fractured rib. They then discuss the two theories proposed to account for arthropathies and spontaneous fractures.

The one theory makes the trophic disturbance of the bones and joints dependent upon atrophy of the cells of the anterior horn; the other refers it to degeneration of the peripheral nerves, especially of those supplying the diseased part. Collecting all available observations, they find that in only four cases alterations in the cells of the anterior horn are reported, and these cases were studied before the development of our present technique.

On the other hand, between 1882 and 1896, seventeen cases have been examined, and in none of these was found alteration of the cells of the anterior horns. In eleven of these cases the peripheral nerves

were examined, and in each case there was nerve degeneration more or less widely distributed, but most marked in the nerves supplying the affected bones or joints. The authors regard the number of observations as yet too few to speak with positiveness, but think the weight of evidence so far in favor of the neuritic, rather than of the myelopathic theory. ALLEN.

149. UEBER NERVENZELLENVERÄNDERUNGEN DES VORDERHORNS BEI TABES (Concerning Changes in the Cells of the Anterior Horns in Tabes). Karl Schaffer (Monatsschrift f. Psychiatrie u. Neurologie, 3, 1898, p. 64).

Schaffer attempts to prove that the cells of the anterior horns of the spinal cord are much altered in cases of tabes with trophic lesions, and that these cellular changes are the cause of the tabetic amyotrophy, osteopathy and arthropathy. In all instances of tabes with trophic lesions reported in the literature by other writers in which the peripheral nerves have been examined, these nerves have been found degenerated, and central lesions, apart from those in Leyden's case, have not been positively observed. Schaffer, however, two years ago published a case of tabetic amyotrophy and arthropathy of one lower limb, in which the cells of the anterior horns of the lumbar cord presented marked chromatolysis. In the present paper he reports the results of an examination of four new cases of tabes, only one of which was without trophic lesions. The changes in the cells of the anterior horns were the same whether amyotrophy, osteopathy or arthropathy existed, and were present in all cases with trophic disturbances. They were more advanced in cases with osteopathy and arthropathy than where amyotrophy existed alone. Central chromatolysis (destruction of the chromophilic elements about the nucleus) was almost invariably the earliest cellular lesion in these cases of tabes, and the disintegration extended peripherally. The edge of the nucleus appeared folded, the nucleus was eccentric, was stained a pale blue (normally it is unstained), and its shape was irregular. Later the nucleus was indistinct, the shape of the nucleolus was much altered, and the cell lost its processes. The nucleus and nucleolus exhibited degenerative changes only when the entire cell was altered.

Schaffer says that the Nissl method, as far as his experience in cellular pathology goes (infectious, toxic, trophic and nutritive changes), does not show specific staining in any case. The lesion is always chromatolysis, which, in its essential features, is always the same. This is a very different opinion from the one he formerly held. In tabes, however, vacuoles in the cells are not numerous, and this infrequency is due to the chronicity of the process, for vacuoles indicate an acute process.

The location of the cellular lesions in the cases of tabes corresponded to the centres of the parts involved in the trophic lesions, and in the one instance in which the latter were not observed distinct cellular changes were absent. He was unable, however, to find distinct groups of degenerated cells, as diseased cells were mingled with normal ones.

In chronic processes, according to Schaffer, only a portion of the cell body is at first affected ("partial degeneration"), and a decrease in the cell's vitality is caused in this way, which manifests itself first at the periphery of the neuron, i. e., in the end ramifications. As this "partial degeneration" increases, more central portions of the neuron are involved, and the alteration of the nerve gradually creeps upward toward the cell body. Chromatolysis must be regarded as a pathological process, and as the result of disturbed cellular nutrition; it is, therefore, an indication of the disturbed function, inasmuch as the latter depends on the cell's impaired vitality.

The tabetic atrophy is due to disease of the cells of the anterior horns, and the latter, in turn, is due to the loss of irritation transmitted through the posterior roots, to a feeble resisting power of these motor cells, and to the influences of the postsyphilitic toxin. The tabetic muscular atrophy resembles that of spinal muscular atrophy, and this fact Schaffer regards as an argument in favor of the spinal nature of the tabetic atrophy. The wasting which occurs in tabes should be looked upon as a complication, and not as an essential part of the disease; the correctness of this view is shown by the comparative rarity of the atrophy and the late appearance of the trophic lesions.

SPILLER.

150. DAS VERHALTEN DER SPINALGANGLIENZELLEN BEI TABES AUF GRUND NISSL'S FÄRBUNG (The Cells of the Spinal Ganglia in Tabes, as Shown by the Nissl Stain). K. Schaffer *Neurologisches Centralblatt*, 17, 1898, p. 2).

The author follows Lenhossék in his classical description of the spinal ganglion cells, and reports upon three cases of Tabes, one of which was examined in the early stages of the disease. By means of the Nissl methods of staining he was unable to find any grave cytological variations from the norm established by Lenhossék. In two of his cases the degeneration was marked in the posterior roots, yet the cells of the ganglia were practically normal, though he notes a quantitative difference in the amount of the coloration of the chromophilic substances, these appearing paler in the cells of the cases showing the stronger grade of degeneration. His results were, therefore, negative, and are distinctly in contrast with those of Marinesco. He furthermore concludes from these cases that the central lesion of tabes is probably not to be found in the spinal ganglia.

JELLIFFE.

151. DIE EMBRYONALEN FASERSYSTEME IN DEN HINTERSTRÄNGEN UND IHRE DEGENERATION BEI DER TABES DORSALIS (The Embryonal Fibre Systems in the Posterior Columns and their Degeneration in Tabes). Trepinski (*Archiv f. Psychiatrie*, 30, 1898, p. 54).

The author here presents, firstly, a careful study of the spinal cord of the newly born child, in which he believes he finds that four methods of development may be present, i. e., the development of the posterior columns, from an embryological point of view, proceeds along four different lines. Some of these embryonal fibre systems are morphologically quite distinct, while others overlap one another, both with reference to their position in the cord and to the time of their development. In accordance with this presentation, he then discusses four cases of tabes, in each of which a different type of degenerative lesion can be demonstrated, corresponding, he believes, with the four embryonal fibre systems demonstrated in his preceding discussion. He states that, bearing these facts in mind, it is readily understood why such a variation in intensity and extension in the pathological pictures may be present. He would further erect at least four types of the disease, according to which of the embryonal fibre systems is most involved. Mixed types are naturally to be expected. The paper is well illustrated and suggestive.

JELLIFFE.

152. ACUTE DEGENERATION OF THE NERVOUS SYSTEM IN DIPHTHERIA. J. J. Thomas (*Boston Med. and Surg. Jour.*, 38, 1898, pp. 76, 97, 123).

In this series of articles an important contribution is made to the pathology of diphtheria which possesses an additional interest from

its relation to the clinical question of death from cardiac failure, which is such a frequent termination of the disease. The early administration of antitoxin would appear to be a practical therapeutic corollary from the constant degeneration of the pneumogastric nerve observed. The writer's conclusions are based upon a series of twenty-five carefully-recorded cases, with autopsy and microscopical examination, from the pathological laboratory of the Boston City Hospital. The specimens were treated by Marchi's method. Other sections were stained by hematoxylin and eosin, and in some of the cases with eosin and Unna's alkaline methylene-blue stain.

The chief change of the nervous system in diphtheria is an acute degenerative process, chiefly parenchymatous, most marked in the peripheral nerves, affecting both the motor and sensory nerves. The myelin sheath is affected first and later also the axis cylinder, generally without infiltration or much multiplication of the nuclei. In this the results correspond pretty closely with those of other investigators, both clinical and experimental, as Prinz, Martin and Crocq, as well as earlier observers, such as Mendel, Leyden and others. Nor do these results stand at marked variance with those of authors who have found changes in the central nervous system, though hemorrhages and myelitis were not found; yet dilatation of the capillaries was observed, and the more marked changes certainly seem possible, though probably rare. The most marked change found in the central nervous system, and that in the brain, as well as the cord, was the presence of fat here, showing that the effect of the toxic substances upon the nerve structures is not confined to the peripheral nerves. These degenerations were diffuse; but, if anything, more marked in the posterior columns of the cord, as has been noted also by Bikeles; a fact difficult of interpretation, unless we assume a less power of resistance to injurious influences of these nerve fibres than is found in others. Indications of the existence of such a fact are not wanting, as shown by the large number of diseases in which degenerative processes have been observed in this region, as for example in pernicious anæmia, pellagra, leprosy and others. Perhaps this affection of the posterior columns may account, as Bikeles suggests, for the ataxia so often observed in diphtheritic paralysis. That the cranial nerves do not always escape the process affecting the nervous system so widely is also certain, as shown by the marked degeneration of the fifth nerve found in one case. These results agree with those of Martin, in that the posterior nerve roots were found fully as much affected as the anterior ones. In regard to the condition of the nerve cells one is not warranted in drawing any very definite conclusions from the results obtained by means of the stains used, but certainly there was no myelitis present. It seems quite probable, however, that some of the nerve cells would show changes, at least in their finer structure, where the nerve processes have suffered so widely.

The processes found in the specimens of heart muscle examined correspond closely to those previously reported by others. There was a parenchymatous degeneration of the muscle fibres, shown by loss of the striations, vacuolization and fatty degeneration. An increase of the muscle nuclei was not present though the nuclei were quite numerous; but this is due to the fact that the hearts examined were from children in whom, from the small size of the fibres, the nuclei appear to be more numerous. The nuclei showed no marked degenerative changes. The dilatation of the vessels was marked, and also the infiltration, both in the myocardium and in the interstitial tissue. This infiltration consisted mostly of small, round lymphoid cells, with a few larger cells, the nature of which could not be accurately determined because of the methods of hardening used—formol, and Müller's fluid. Certainly, leucocytes played no important part in the process. The

most constant change was the interstitial process. The fact that changes were found, more or less marked, in all the pneumogastric nerves examined, seems to point to considerable influence upon the mode of death in the cases of sudden death. In what way does the poison act? It seems most probable that in such cases it acts through the nerve structures, interfering with their normal function, and that this may occur before degenerative processes have proceeded far does not lessen in any way the importance of their occurrence, but would rather lead us to place greater weight upon slight changes, where other obvious causes of death, as markedly degenerated heart muscle, do not exist. The variability of the amount of changes, both in the heart muscle and in the nerves, may point to a varying cause for these cases of sudden death; but the argument of the disturbance of the functions of the nerve seems strongest.

To sum up, the changes in the nervous system produced by diphtheria are: (1) a marked parenchymatous degeneration of the peripheral nerves, sometimes accompanied by an interstitial process, and hyperæmia and hemorrhages; (2) acute, diffuse, parenchymatous degeneration of the nerve fibres of the cord and brain; (3) no changes, or but slight ones, in the nerve cells; (4) acute, parenchymatous and interstitial changes in the muscles, especially the heart muscle; (5) occasional hyperæmia, or infiltration, or hemorrhage in the brain or cord, in rare cases severe enough to produce permanent troubles, such as the cases of multiple sclerosis or of hemiplegia which have been observed. Finally, the probability that the cases of sudden death from heart failure in diphtheria during the disease, or convalescence, are due to the effects of toxic substances produced in the disease upon the nerve structures of the heart.

SHIVELY.

CLINICAL NEUROLOGY.

153. *TABES AVEC CONSERVATION DES REFLEXES ROTULIENS* (Tabes with Preserved Patellar Reflexes). MM. Achard and Lévi (La Med. Moderne, 9, 1898, p. 176).

A typical case of tabes, except for the preservation of the knee-jerks, is reported, with the autopsy. There was sclerosis of the posterior columns, most marked in the sacral and cervical regions. The cornu-commissural zone, the comma-shaped columns of Schultze, and the other "descending areas" were well preserved. At the junction of the lumbar and dorsal segments Westphal's zone of entry of the posterior roots ("Wurzel-Eintritt") was observed to be intact. Numerous cases have now been reported of various forms of disorder of the spinal cord, in which it might be expected that the knee-jerk would be lost, but in which it has persisted, and wherever careful post-mortem investigation has been made this zone has been found intact. Lehmann, Kraus, Westphal, Minor, Pick and others have observed this persistence of the reflex in cases of tabes and "combined disease," and accounted for the fact by the integrity of this area in the cord.

MITCHELL.

154. *TABES DORSALIS UND WANDERNIERE* (Tabes Dorsalis and Floating Kidneys). A. Habel (Centralblatt für Innere Medicine, 18, 1897, p. 161).

Attention is called to the fact that in cases of tabes a large proportion of wandering kidneys was observed. Thus in the Zurich Clinic some 14 per cent. of all the cases of tabes presented this anomaly, and in the case of the women some 25 per cent. were noted. The proportion observed in the general medical clinic at the same institution for the same time being 1 per cent. The author believes it not improbable that some sort of causative or predisposing condition may exist in this disease or in this anomaly.

JELLIFFE.

155. ARTHROPATHIES TABÉTIQUES (Tabetic Arthropathies). M. Hirtz (La Med. Moderne, 9, 1898, p. 48).

The author reports a case presenting a joint lesion of tabes in the unusual situation of the metatarso-phalangeal articulations. Radiographs were shown, demonstrating the condition excellently.

MITCHELL.

156. DES TROUBLES DU GOUT ET DE L'ODORAT DANS LE TABES. (Disturbances of Taste and Smell in Tabes). M. Klippel (Archiv. de Neurologie, 3, 1897, p. 257).

The author shows that disturbances in taste and smell are by no means as infrequent as a search of neurological literature would lead one to suppose, and, moreover, that such affections are of much value, in that they may be found among the earlier symptoms of this disease. The symptoms noted are various and often difficult of exact observation. They consist in paraesthetic sensations in the nose, loss of smell and taste, disturbances in the ordinary sensibility, perversion of smell and taste, and in some cases taste or smell crises may be observed, those in the nose being associated constantly with prickling, and culminating in violent and repeated sneezing. In the severer grades of these sensory disturbances accompanying involvement of other cranial nerves is often observed. In one case reported by the writer an autopsy was performed, and microscopical examination of the olfactory, glossopharyngeal and trigeminal nerves and their corresponding ganglia showed marked degenerative changes, thus accounting for the symptoms.

JELLIFFE.

157. ON PERIODIC VOMITING IN TABES; GASTRIC CRISES. P. Ostankow (Obosrenie psichiatric (Russian), 1897, Nos. 7 and 8; Neurol. Centralblatt).

The author reports upon two cases of gastric crises. In the first case the gastric symptoms lasted for many weeks, with intervals of freedom of from three to four days at a time. In the second case the crises were shorter, but were almost uninterrupted. In both of the cases there was a prodromal period, characterized by a loss of sleep, retention of the urine, restlessness and anorexia. In the first case during the crises the pulse was increased in frequency; in the second there was arrhythmia and interrupted variation in tension. Cerium oxalate in doses of from .05 to .15 gm. (1 to 3 grs.) t.i. d. gave relief in both cases.

VOGEL.

158. NOTE SUR LA RETOUR DE LA SENSIBILITÉ TESTICULAIRE DANS LA "TABES" (Note on the Return of the Sensibility of the Testicle in Tabes). E. Bitot et J. Sabrazes (Revue de Med., 17, 1897, p. 156).

Pitré first noticed that analgesia of the testicle was a nearly constant sign in tabes, occurring in as many as 75 per cent. of the cases. The authors hold that this analgesia is of interest in that it does not seem to follow the same rules as other sensory changes in tabes, relative to its constancy after once having been established. They report three cases in which the sensibility returned, and in two of them there was a well-marked gain in the sexual vigor of the patients.

JELLIFFE.

159. ON THE EARLY AND LITTLE KNOWN SYMPTOMS OF TABES. W. Bechterew (Revue de Psych. (Russian), 1897, No. 8; Rev. Neurologique).

The author calls attention to the preservation of the reflexes, cutaneous abdominal and epigastric, coincident with the loss of the

tendon reflexes in the initial stages of tabes. He also speaks of the analgesia of the popliteal nerve in the popliteal fossa as a sign more frequent and more constant than Biernacki's analgesia of the ulnar nerve or Sarbo's analgesia of the peroneal. VOGEL.

160. A CASE OF PSUEDO-TABES FOLLOWING DIPHTHERITIC INFECTION IN THE PENIS. J. W. Courtney (Atlantic Medical Week, 9, 1898, p. 33).

The author cites an interesting case in which there was a typical diphtheritic ulcer on the penis of a man of 47 years of age. Similar ulcers were found upon the ring finger of the right hand. About a month after healing the patient noted a loss of power in his limbs as he arose, and also a numbness in the left heel. Later his gait became unsteady, and he had beginning paralysis of accommodation. At the time of examination the patient had an extreme ataxic gait, marked Romberg, pupils equal and normal. No cranial nerve palsy, right hand weak, incoordination. Lower extremities weak and atrophied muscles, knee jerks absent, no ankle clonus, no loss of sensation and no marked electrical disturbances.

On tonic treatment the patient gradually improved, and the prognosis seemed favorable for a complete recovery. VOGEL.

THERAPY.

161. L'ÉLONGATION VRAIE DE LA MOELLE ÉPINIÈRE ET SON APPLICATION AU TRAITEMENT DE L'ATAXIE LOCOMOTRICE; RECHERCHES EXPÉRIMENTALES ET THÉRAPEUTIQUES (Locomotor Ataxia Treated by Stretching in the Sitting Position. MM. Gilles de la Tourette et A. Chipault (Gaz. Hebd. de Med. et Chir., 2, 1897, p. 491).

The authors reported some observations upon the topographical anatomy of the spinal cord which convinced them that it was possible for certain well-defined manœuvres to make decided elongation of the cord, and that these could not practically be done by suspension; but by passive flexion of the body with the patient seated with the legs extended, there could be an elongation of about one centimetre, the stretching affecting the posterior portion of the column to the level of the first lumbar pair of nerves.

With an apparatus constructed for the purpose, they have experimented on ten healthy individuals, who were competent to render a report of their sensations, and upon 47 ataxics, all of whom were in the second stage of the disease. Only ten of the patients treated did not seem to receive any benefit; 22 were much improved, and 15 were benefited. MITCHELL.

162. LE TRAITEMENT DE L'ATAXIE LOCOMOTRICE PAR L'ÉLONGATION VRAIE DE LA MOELLE ÉPINIÈRE (Treatment of Locomotor Ataxia by True Stretching of the Spinal Cord). M. Gilles de la Tourette (Gazette des Hôpitaux, 70, 1897, p. 1,368).

The good results of this therapeutic method, which have already been presented to the Academy by M. Chipault have been confirmed by further experience. In collaboration with M. Gasue a large number of tabetics were observed at the Salpêtrière, upon whom the treatment was regularly carried out for a sufficiently long period to test the method. Seventeen cases out of twenty-one were considerably benefited, especially as regards pain, genito-urinary symptoms (excepting incontinence, which was little affected) and incoordination. This percentage will doubtless appear large, but it should be remembered that not all the cases of tabes were indiscriminately submitted

to the stretching treatment. This treatment ought to be refused to greatly debilitated subjects, to those in whom the disease is pursuing a very mild course, in arthritic cases and to cases in whom laryngeal crises occur. It should, moreover, be determined beforehand that the vertebral column is neither too flexible nor too rigid; a sensation of numbness felt in the feet during the application of treatment is the best proof that elongation is actually produced and that the apparatus has been properly applied. SHIVELY.

163. TRAITEMENT DE L'ATAXIE DES TABÉTIQUES PAR LA MÉTHODE DE REÉDUCATION—MÉTHODE DE FRENKEL (Treatment of Tabetic Ataxia by Reéducation (Method of Frenkel). Maurice Faure (Presse Medical, 5, 1897, p. 352).

The author records the results of the application of systematic coördinated exercises upon 13 cases in the service of Prof. Raymond at the Salpêtrière. In all, the application of Frenkel's method was coincident with an improvement of the ataxia, the more pronounced the more severe the condition had been. He describes the movements employed and discusses the general principles to be observed. The seances should be short in the beginning of the treatment; later they may be longer, though it is advisable to allow a brief rest every ten minutes. It is important that some one with sufficient authority and intelligence should be present to control the movements of the patient. No movements should be employed that require strength, the idea being to develop dexterity, and it must be remembered that the extreme laxity of the muscles does not limit the movements of the joints, as in a normal person, and that the bones are frequently fragile. Altogether, 40 cases have been reported by various authors, with 40 successes. The object of this method is to improve the ataxia. It is of no value against the other symptoms, and should be used only in those cases in which coördination is pronounced. The contraindications are rapidly developing ataxia, grave general symptoms, either trophic or visceral, particularly if there is any cachexia, amaurosis or psychical disturbance, accompanied by paralysis, although true paralysis must not be confounded with the apparent forms resulting from extreme ataxia; cases accompanied with hyperæsthesia, in which fatigue appears rapidly, and, finally, the presence of arthropathies or fractures. The most favorable cases are those in which the ataxia develops rapidly at first, or those in which it is almost the only symptom. The results will, of course, be better if the patient is young, energetic and intelligent. In the beginning the benefit is usually very pronounced. This is due largely to suggestion. Later improvement is more gradual, and, finally, there comes a time when the patient is not further benefited, and treatment should be suspended. SAILER.

164. TRAITEMENT DE L'ATAXIE DANS LE TABES DORSALIS PAR LE REÉDUCATION DES MOUVEMENT (Méthode de Frenkel). Hirschberg (Archiv. de Neurologie, 2, 1896, p. 337).

The author presents his results with Frenkel's methods on nine cases treated, going greatly into detail as to the precise series of movements practised. As a rule, he began with 30-minute exercises, and extended them to an hour, rarely exceeding that limit, and always ceasing the moment that muscle tire became evident. In all of his cases there was some improvement, though three of them had grave ataxia, whereas the others had but the early symptoms. The improvement was evident, not only with the walking, but also with the feeling of the patient, and the author believes that this method is of service in all stages of tabes, and is, perhaps, contraindicated only when the disease is progressing rapidly. VOGEL.

Book Reviews.

THE GENESIS AND DISSOLUTION OF THE FACULTY OF SPEECH. A Clinical and Psychological Study of Aphasia, by Joseph Collins, M. D., Professor of Diseases of the Mind and Nervous System in the New York Post-Graduate Medical School, etc. Awarded the Alvarenga Prize of the College of Physicians of Philadelphia, 1897. New York: The MacMillan Company, 1898.

A book that has received a prize is like a painting marked, "Hors concours." You are told thereby that the artist has done good work, and that there must be merit in his production. If you cannot see it, the fault is with your powers of observation and not with the painting. In this instance we are ready to endorse the verdict of the jury. Dr. Collins' essay on aphasia was altogether worthy of the prize, for it is characterized by an intelligent grasp of the subject, by much originality of thought, and by a critical spirit which has enabled the author to give a just estimate of contending theories. He has laid down his monograph on the broadest lines, including under aphasia "the total inability, or partial disability, of an individual to make outward expression of thoughts, feelings, or other states of consciousness, whether such disability result from interference with the formation of the mental content, or in the emission of it."

The subject is subdivided (in the introductory chapter) into: 1. True Aphasia—aphasia of apperception, due to lesion of the speech region; 2. Sensory Aphasia; 3. Motor Aphasia; 4. Compound Aphasia. The distinction between true aphasia and motor and sensory aphasia is not "posited" (to use one of the author's many pet words) very clearly, nor is it maintained throughout the book; and, surely, motor aphasia is due to a lesion of the centre itself, as well as to a lesion of the "motor pathways over which motor impulses travel in passing to the peripheral speech musculature."

In the chapter on the history of aphasia (an unusually well written chapter) the author gives an excellent and interesting account of the writings of previous authors. We have no fault to find with his estimate of the relative importance of the contributions by various standard writers, but regret that he did not enter more fully into the analysis of the essays by Hughlings Jackson, which, to our thinking, have been the most enduring contributions to the psychology of aphasia. To be sure, the author states that "three great names . . . stand out above those of all others (aside from Broca and Wernicke), namely, Trousseau, Hughlings Jackson and Kussmaul."

Chapters 3 and 4 are devoted to the genesis and functions of speech and to the conception of aphasia. In the latter the author defines his position in accord with those who believe speech to be the function not of any one or two centres, but of the zone of language in which the special centres and the association tracts connecting them are situated. It is a relief to know that the speech area is gondola-shaped. We had thought it resembled a slipper, but gondola it is. Flechsig's recent publications have formed an important support for the author's conception of aphasia. He has accepted the professor's theories in toto, and we fear that he has not read them with the same "criticalness" which he has applied to the writings of others. Collins does not believe in a special graphic

motor centre, and his arguments on this point are convincing. We cannot approve entirely, however, of his objections to the form of subcortical motor aphasia, and think Proust-Lichtheim's test is more satisfactory than the author seems willing to believe.

The chapters on sensory and subcortical sensory aphasia contain comprehensive statements of the questions involved. The clinical and pathological data furnished by the writer help to make these chapters particularly valuable. We cannot refer in detail to the many interesting points discussed, but may note in passing that reference is made to the case of Freund, which shows "that the peripheral auditory neuron can be normal for the conduction of ordinary sounds, and diseased for the conduction of sounds having highly differentiated significance."

In the chapter on total aphasia the author has shown the results of his own clinical experience. Many other writers have erred in defining their cases always as belonging to the type either of motor or of sensory aphasia, and in not allowing that the two may be combined. During the period of convalescence these mixed or "total" aphasias are most puzzling.

Of the remaining chapters of the book it need only be said that Collins has done ample justice to the consideration of the etiology, the morbid anatomy and the treatment of aphasia, but the reader must remain impressed with the fact that in nine cases out of ten the aphasia is only one of a series of symptoms. In the chapter on treatment, pedagogic methods are properly extolled.

The attentive reader will lay down this book impressed by its matter and its style. As we have said above, there is much originality in the former, and we cannot help saying that there is still more in the latter. Few medical men of our day wield as facile a pen, but there is danger in this ease of diction—the danger that the reader's attention will be arrested first by the style, and lastly by the contents. Collins has a special fondness for archaic terms and for new (composite) terms. Where is the "word coining" centre that harbors "awakement," "paronymization" (shades of Wilder!), "dextrality," "hostilely," "ideate," "materies of storage," "undislodgable?" There is every reason to believe that the editor of the next Century Dictionary will have a serious task before him. But, to be original is meritorious, and many of us will ascribe our "awakement" on the subject of aphasia to the "materies" of Collins' splendid monograph.

B. SACHS.

A TEXT BOOK OF THE PRACTICE OF MEDICINE. By James M. Anders, M.D., Ph.D., LL.D., Professor of Practice of Medicine and of Clinical Medicine in the Medico-Chirurgical College of Philadelphia. W. B. Saunders. Philadelphia, 1898.

Dr. Anders has written quite a large and presentable work on the practice of medicine. In it about 200 pages are devoted to the diseases of the nervous system. In a practice of medicine one does not expect to find an extensive treatment of any one particular field, and hence the chapters on nervous diseases will be of little service to the neurologist, but to the beginning student of neurology we believe the chapters in question will be of a great deal of service. The style is good, the information, for the most part, accurate, and there is one feature commendable from the pedagogic point of view. This consists in the large number of tables of differential diagnosis, specially valuable for the beginner, though the specialist realizes their inadequacy.

VOGEL.

THE PSYCHOLOGY OF SUGGESTION. A Research into the Sub-conscious Nature of Man and Society. By Boris Sidis, M.A., Ph.D. Appleton and Company. 1898.

So much has been written upon the general allied subjects of hypnotism, unconscious cerebration and suggestion, which for the most part serves the purpose of mental obfuscation, that it is a relief to read a book in which an endeavor has been made to state the problems in a clear and comprehensive manner. This, we believe, the present volume does in the main. In three parts "Suggestibility," "The Self" and "Society" are treated. The opening chapters are the best. Here the author, from observation and a series of experiments, formulates a number of laws with reference to what he terms normal and abnormal suggestibility. "Normal suggestibility," he states, as one of these laws, "varies as indirect suggestion and inversely as direct suggestion." Its opposite, the law of "abnormal suggestibility." While these laws look attractive, and the small series of experiments quoted would point to their general application, we cannot help but feel that the author has very indistinctly differentiated what he means by direct and indirect suggestion, and certainly we are not prepared to accept his dicta on the evidence here presented. In our opinion, Baldwin's presentation of much the same subject is far more reasonable and scientific, from the psychological point of view, and his conclusions would be far from those expressed in this volume.

The author's point of view is well illustrated in his encomium of Gurney's classification of the hypnotic phenomena, which he designates as the most philosophical, and hence the best. Thus, he would ally himself rather with those working purely in the field of the Psychical Research Society, commendable though their workers may be in many respects, with their fallacious evidence derived from the so-called "law of probabilities," rather than with the more conservative, and, we believe, more careful "physiological psychologists."

In Part 2, "The Self," we find most of the familiar anecdotes which are repeated from book to book, each author using them as illustrations of his own point of view, though often the points of view are diametrically opposed. Psychical phenomena seem to be more than usually elastic from an interpretative point of view.

The author apparently adopts Binet's exposition of the "double personality," and cites many of the threadbare examples, and adds an interesting one of his own.

In Part 3 "Society" is dealt with, and the familiar caption of the "suggestibility of the mob" is treated. The facts brought together are extremely interesting, but we do not think that the analysis of the conditions is thorough nor satisfactory. Impressionists are rarely scientists, and a general, hazy delineation of a large movement, termed mania or epidemic for descriptive effect, which involves a great many factors, is not a careful presentation of the principles of social movements.

Considered as a whole, the work is instructive and suggestive; it is a little too anecdotal in character, a feature of all the works of this kind, and somewhat hastily constructed, but well worth the reading.

VOGEL.

THE PROCEEDINGS OF THE AMERICAN MEDICO-PSYCHOLOGICAL ASSOCIATION. Fifty-third Annual Meeting held in Baltimore, May 11th-14th, 1897.

The present volume, like most of the preceding numbers, is made up in the main from reprints of articles which have appeared in the "American Journal of Insanity." The wide scope and the high character of the work are an evidence of the activity of the society and the scientific interests of its members.

JELLIFFE.

THE
Journal
OF
Nervous and Mental Disease

AMERICAN NEUROLOGICAL ASSOCIATION.

Twenty-fourth Annual Meeting, held at The New York Academy of Medicine, New York, May 26th, 27th and 28th, 1898.

The President, Dr. Graeme M. Hammond, in the chair.

PRESIDENT'S ADDRESS.¹

Gentlemen: Before proceeding to say the few words which will constitute my address to you, I desire to express my warmest thanks for the honor you have conferred upon me in choosing me your President for this year. Appreciating, as I do, that the Presidency of the American Neurological Association is the highest honor the neurologists of this country can bestow, I cannot but feel gratified and complimented that in your eyes I am considered worthy of it.

I will not detain you with many words. Even were I disposed to do so, the unprecedented number of papers on the programme, more than have ever been presented in the history of this association, compels me to leave unsaid the words I had originally intended should be your greeting.

I cannot refrain, however, from calling your attention to certain matters which seem to warrant your earnest consideration.

One of the charter members of this association, one who was for many years your faithful secretary and treasurer, and whose constant adherence and earnest endeavors

¹Read at the twenty-fourth annual meeting of the American Neurological Association, May, 1898.

have tended to place this association on the sound scientific foundation on which it stands to-day, has passed away. I refer to Dr. E. C. Seguin, who, as a pure scientist, helped in no inconsiderable way to advance the knowledge of neurology in America. The history of American neurology cannot be written without the name of Dr. Seguin on the title-page. It must be a consolation to you all to remember that you recognized his years of service as an American neurologist by electing him to honorary membership in the association, the greatest honor the association can confer, an honor which you may know gratified him in his last days, and made him feel that his life-work was appreciated by those who were capable of understanding him and his works. It will give me pleasure, at the proper time, to appoint a committee to draft suitable resolutions, expressing our sorrow and regret at the loss of a most valued member, to whom neurological science was deeply indebted.

Almost from our beginning we have had classes of membership known as associate and honorary members. In the beginning we were weak, certainly as a national body, if as nothing else. The possession of associate and honorary members of foreigners who had achieved greatness in neurology seemed to make us more important, and to reflect some of their greatness upon us; thus, by reflection, making us seem greater as an association than we really were. Many of these were elected, not only without solicitation on their part, but sometimes without even their consent. In the great majority of instances they have never taken the slightest notice of their election. They neither add, except in the most rare instances, to the scientific work of our association, nor do they by their names attract new members.

This association of American neurologists is to-day capable by its works of maintaining a prestige in the neurological world without depending upon the names of those who neither, apparently, desire to be affiliated

with us, nor show a just feeling of reciprocity by electing American neurologists to membership in their societies. This association should be what its name stands for — an association of American neurologists. It is so actually. It has never received either aid, encouragement or contributions, except reprints, from its foreign members. They do not want us. We do not need them. I would, therefore, recommend such legislative action by the association as either to abolish associate membership, or, if that seems inexpedient, to prevent the addition of new members to that rank.

It is the same in regard to honorary membership. It seems to me as if honorary membership should be conferred only upon members of the association, who, either by long years of faithful service, or by their scientific reputation, have achieved the right to the distinction and honor of being elected to honorary membership in this association.

I do not mean, for a moment, to imply that we are not honored by our honorary membership as it stands to-day. The fact that one is elected to honorary membership is a sufficient warrant of distinction in itself. Neither do I desire to be considered narrow-minded in arguing for this policy of exclusion, but I would prefer to see this association American, and American only. I now declare the scientific session open.

NOTE.—By the unanimous vote of the Association the following distinguished physicians were elected Honorary Members:

Sir William R. Gowers, of London, England.

Dr. Byrom Bramwell, of Edinburgh, Scotland.

Prof. H. Nothnagel, of Vienna, Austria.

Original Articles.

ON MYOTONIA.¹

BY GEORGE W. JACOBY, M. D.

The disease, first known through the description of the Silesian physician, Dr. Thomsen, and since then called after him, is an affection scientifically so remarkable and interesting that I may be pardoned for again bringing this subject before you. Nor would I do so now, were it simply for the purpose of recording another case of Thomsen's disease (myotonia congenita); for, while the affection must be one of infrequent occurrence, scarcely more than 50 cases having been published, yet all of these cases resemble each other to such an extent, that one typical case may fittingly be looked upon as a paradigm of the rest.

The subject must, however, to-day be looked upon from a broader point of view than that of mere casuistics.

The history and literature of Thomsen's disease up to 1886 will be found in Erb's monograph, that from 1886 to 1889 in his article in the *Deutsches Archiv*, and that from 1889 to 1894 in the article by Suesskand, published in the *Zeitschrift für klinische Medizin*, vol. xxv. The only case of unusual interest published since then is the one with autopsy by Dejerine and Sottas, and to this I shall have occasion to refer again.

Our knowledge of this affection is now so well defined that the term Thomsen's disease, or myotonia congenita, embraces a clearly circumscribed clinical entity,

¹ Read before the American Neurological Association, May 27th, 1898.

and constitutes a picture to which nothing can possibly be added; nevertheless, it seems to me that the time has come when we must limit this name to the actually congenital cases, and not expand it to include the acquired ones, or, possibly, even such as have the myotonic disorder and myotonic reaction, but in other respects differ to a greater or less extent from cases of Thomsen's disease. In view of the cases which I herewith publish, I think it proper to assign all such cases to the one clinical category of myotonia, and to then subdivide this class into various groups.

If we analyze the well-known picture of the disease, we find it made up of the following components:

1. *The Etiology.* Here the most important factor is, beyond a doubt, heredity, either as a direct transfer from the ascendant, or only indirectly by inherited disposition; atavistically, as in Weichmann's case, or where collateral branches are affected, as in the cases of Knud Pontopidan and Bernhardt; or the disease may occur as a family type, without direct heredity.

2. *The myotonic disorder of movement;* i. e., the occurrence of tension, stiffness and tonic spasm in the voluntary muscles at the beginning of intended movements.

3. *The myotonic reaction,* which is made up of normal mechanical, faradic and galvanic excitability of the motor nerves, and an increased mechanical, faradic and galvanic excitability of the muscles. Here with the galvanic current only closure contractions are obtainable, and these are as strong with the anode as with the kathode; the contractions are always slow, tonic and prolonged.

In many muscles strong faradic currents produce irregular undulating contractions and stabile galvanic currents, rhythmical contraction waves which follow one upon the other.

3. *Hypertrophy of the muscles.*

4. *Absence of all symptoms pointing to gross involvement of the nervous system.*

Of the published cases of myotonia congenita, the large majority correspond absolutely to these requirements. Cases which show symptoms of organic disease of the central nervous system, with myotonic disorder, but without myotonic reaction, such as Dana's, can at once be excluded from the group of myotonias. A large minority, however, show certain smaller deviations from the typical picture. Aside from variations in intensity and extensity of the disease, all or only some muscles being severely or slightly affected, the most frequent deviation consists in the affection being neither hereditary nor congenital. Such cases have been described by Seligmüller, Peters, Weichmann, Rieder, Vigoroux, G. Fischer, Erb and Suesskand.

The following history is that of a typical acquired case:

Case I.—W. J. G., of Wheeling, W. Va., was referred to me by Dr. J. Schwinn of that city, with the diagnosis Thomsen's disease, and was presented at the New York Neurological Society on Nov. 2d, 1897, as a typical case of this disease.

Patient is 28 years of age, was born in Ireland, and came to the United States in 1884. He is a railroad brakeman by occupation.

Family History.—His grandparents lived to an old age. His father died at the age of 70, cause unknown; his mother is living and well at the age of 69. She had nine children, of which two are dead. The others, five sisters and one brother, are healthy.

The patient himself was rather delicate up to his 14th year; otherwise, with the exception of an attack of measles, and one of whooping cough, he was perfectly well. He never noticed any difference between himself and his schoolmates in regard to physical strength, and was able to participate in all out-of-door sports and games. Psychically, he says he was rather timid, and perhaps slightly backward. At the age of 18 he had an attack of typhoid fever without complications, which kept him in the house for five weeks. When he went out for the first time, he experienced a severe painless cramp in the calves of both legs, more marked on the right side. This cramp lasted about two minutes. During the next two weeks it recurred several times each day, and his legs felt somewhat weak. He then complained of feeble sexual powers with frequent nocturnal emissions, but was well and gained rapidly in weight

and strength. During this time his muscles increased in size, so that they seemed to him unusually large, and now that he is questioned, he thinks he was somewhat stiff during all this time.

About two years after this attack of typhoid, he began to complain of stiffness of his legs, especially noticeable when climbing a ladder, or stepping into a car. He also noticed that after a prolonged rest the stiffness in his muscles was always decidedly increased, and that after the first few moments the stiffness gradually disappeared and he again had full use of his limbs. At first only the muscles of his legs were affected, and these slightly, then gradually nearly all of the voluntary muscles became involved.

During the time of this progression, and before his arms were seriously affected, his legs became so bad that in 1893, when he jumped from a car, the muscles would cramp up so that he would fall; then he could get up only by using his hands as a support. If in walking or running he stubbed his toe, his leg would stiffen so that he could not lift his foot from the ground. It would, as he expresses it, "stick to the earth," and then he would fall. Since a year he has complained of a similar condition of stiffness in his hands, so that when turning the wheel of the car brake, it would require a long time before he could loosen his grip and straighten his fingers. This was accompanied by a feeling of tension and stiffness in the arm; also when his arm was forcibly extended he could not bend it again until the spasm ceased.

Recently the neck muscles, the face muscles and tongue have become affected, though not to so marked a degree. He also feels a "stiffness" when he closes his eyes, and at times he feels his right eye "catch" when he turns his eye outward. He has no pains and feels perfectly well. The trouble is worse at certain times than at others; some days he seems all right, but he does not think that the weather has any influence upon his condition. He has often wondered at the fact that though his muscles seemed to increase a great deal in size, his strength was not correspondingly great, in fact, he thinks he has become weaker.

Status.—Medium height. Panniculus adiposus poorly developed, in contrast to the muscular development which is extreme, giving the man the appearance of an athlete. Though all the muscles show an exceedingly strong development, the muscles of the legs, forearms and arms are especially large. The calves, when contracted, measure 42 cm. in circumference at their largest part.

The accompanying photograph gives a good illustration of their appearance. The gross power of the muscles is feeble in comparison to their size. Aside from the muscular disorder,

no symptom of disease of any organ can be discovered. Sensory disturbances are absent; the superficial reflexes are normal. Triceps and Achilles tendon reflexes are not obtainable. The knee-jerk is very well marked, in the beginning even exaggerated, but becomes exhausted after repeated blows, so that at the end of a prolonged examination it is no more

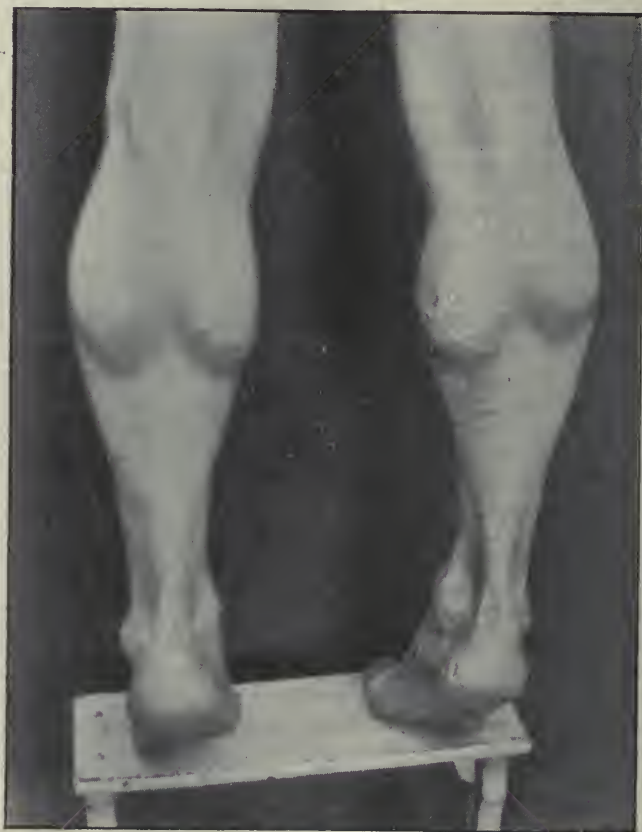


FIG. I.

obtainable, reappearing, however, in its increased state after a rest of half an hour or more.

Very pronounced is the myotonic disorder of all the muscles. When the lids are firmly pressed together, they cannot be opened at once, although he makes great effort to do so. Only after a time is he able to open his eyes. The spasm in

these, as well as in all other muscles, lasts from 15 to 25 seconds.

The internus of the right eye seems somewhat affected, inasmuch as the eyeball cannot at times be brought to the inner canthus or at times having been brought to that position, cannot promptly be returned to its normal position of rest.

The pupils react promptly to light and accommodation, the visual field is not restricted and the fundus is normal.

This condition of spasm is present in the tongue and in the muscles of mastication.

In the tongue, however, we find that the spasm occurs only when the intrinsic muscles are exerted, but that the genioglossus is free, inasmuch as no spasm occurs in protrusion of the tongue. So also the disorder of the masticatory muscles is limited to the masseters, while the pterygoids (lateral movements) are free. The masseters stand out hard and rigid like bars of iron, when the teeth are forcibly closed. The spasm, contraction and after-duration, is also present in the muscles of the face, but it is not so marked as elsewhere.

In the movements of the arms, hands, and legs, this myotonic disorder is most apparent. This is particularly noticeable in the hands when any object is tightly grasped, and in the legs in going up stairs or in jumping from a low stool.

The thorax muscles are implicated with excessive movements; thus when he expands his chest to its maximum capacity, he is not at once able to empty it, but must allow several seconds to elapse. The muscles when uncontracted convey no different sensation to the examiner's hand than does a normal muscle.

Mechanical Excitability.—The mechanical excitability of the trunks of the motor nerves (n. facialis, plexus brachialis, n. ulnaris, n. peroneus) is not increased.

Mechanical Excitation of the Muscles.—All the muscles showed the formation of the marked lasting furrows, upon excitation with the percussion hammer. When stronger blows were employed, an idiomuscular mound was formed in certain muscles (biceps, pectoralis) which remained so long and was of such consistency, that it could be distinctly palpated and manipulated. The total contractions of the muscles showed no after-duration, were very marked upon the first blow, then with each succeeding blow grew less and less, until finally they could no longer be obtained. Even when they were thus lost, the local and fibrillar contractions were obtainable to their maximum extent.

The electrical examination showed normal faradic and galvanic excitability of the motor nerves. The muscles with medium faradic currents showed slow, lazy, lasting contractions (20 to 25 seconds). Single opening shocks, no matter how strong, produced only short quick contractions.

The muscles showed certain quantitative and qualitative changes to the galvanic current. In nearly all of the muscles a contraction could be obtained with less current than is normally the case; for instance, in the triceps a Ka. C. C. is produced with $\frac{1}{2}$ M. A. of current. An C. C. is obtained with about the same amount of current as Ka. C. C., or in some muscles with a very little more. Even with minimum currents the An. C. C. shows a certain amount of slowness; as soon as the current is increased, both contractions, Ka. C. C. and AN. C. C. show marked slowness, tonicity and after-duration.

In this patient I was also, after various attempts, able, during the passage of a strong steady galvanic current, to obtain the peculiar rhythmical contraction waves as described by Erb; a large flat electrode being fastened to the back of the shoulder, the other electrode placed in the palm of the hand or upon the flexors of the forearm, and the current of at least 20 M. A. closed. The primary result is a lasting tonic contraction of all the muscles of the arm. Then after about half a minute, slow wavy contractions set up and pass from the Ka. to the An, one wave following another at intervals of 1 to 2 seconds. In the lower extremities I was also able to obtain these waves, but could not satisfy myself of their direction from the Ka. to the An. These waves were not always demonstrable. Their production always required strong currents and a great deal of manipulation of current and electrodes.

Examination of the blood showed nothing abnormal. Hæmoglobin, 80 per cent.; red blood corpuscles, 4,360,000; white blood corpuscles, 7,450.

For purposes of microscopical examination two pieces of muscle were excised from the left quadriceps, and one piece from the left biceps. These pieces were kindly prepared for me by Dr. F. Schwyzer.

Immediately after excision each piece of muscle, in order so far as possible to prevent permanent shortening, was fully extended and thus fixed by means of a skewer, both ends of which were pointed. Each end of the muscle piece was slipped over the corresponding end of the skewer, and thus extended, the pieces were placed for hardening in a mixture consisting of formalin 1 part to 4 parts of physiological salt solution; they were then transferred to alcohol, finally embedded in celloidin, cut and stained. When the pieces were transferred from the formalin to the alcohol, it was noted that one of them had torn away from its fastenings and was very much contracted, while the others remained fastened and extended. This accident, as will be seen later, proved to be rather fortunate. The specimens were stained with iron hæmatoxylin (nuclear stains), and after preparation were compared with specimens taken from normal muscle and with others from myotonia congenita.

A careful examination of all the specimens showed that we had two distinct pictures before us, both of the transverse as well as of the longitudinal sections.

In the one transverse picture (Fig. II.) we found muscle fibres more or less polygonal in shape, the nuclei in a few of the fibres somewhat increased in number, and the interstitial tissue also somewhat augmented. The diameters of the fibres in these sections were as follows: In 100 fibres, the smallest fibre meas-

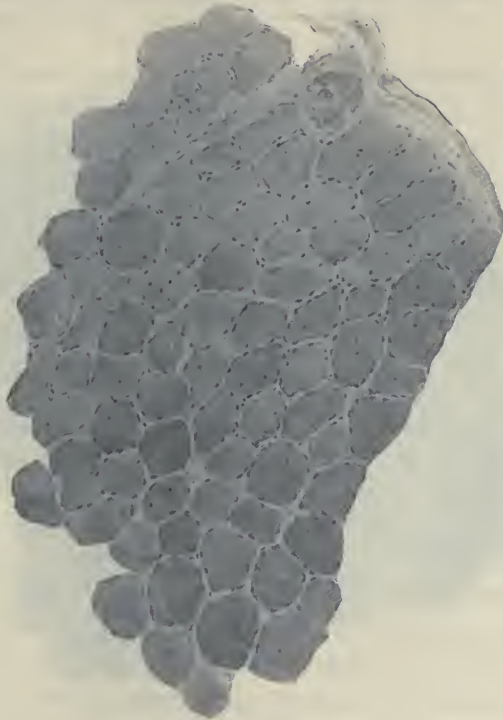


FIG. II.

Transverse section of extended muscle. (Oc. 4., Obj. A. Zeiss.)

ured 60 microns; the largest, 100 microns. Of these 100 fibres, 90 per cent. measured less than 90 microns, and only 10 per cent. were found to measure between 90 and 100.

In the second series (Fig. III.) of transverse pictures we find the single fibres mostly with rounded edges, the nuclei and interstitial tissue decidedly increased, and almost all of the fibres very much larger than in the other specimens. Meas-

urements here, again of 100 fibres, showed the smallest to measure 75 microns; the largest, 195 microns in diameter. 33 per cent. measured between 75 and 100; 60 per cent. between 100 and 150 microns, and 71 per cent. between 150 and 195 microns. The longitudinal sections again revealed the same variations in different sections, some showing the same characteristics as those of the second series of transverse sections, others not.

In addition it was seen that in the one set all the muscle

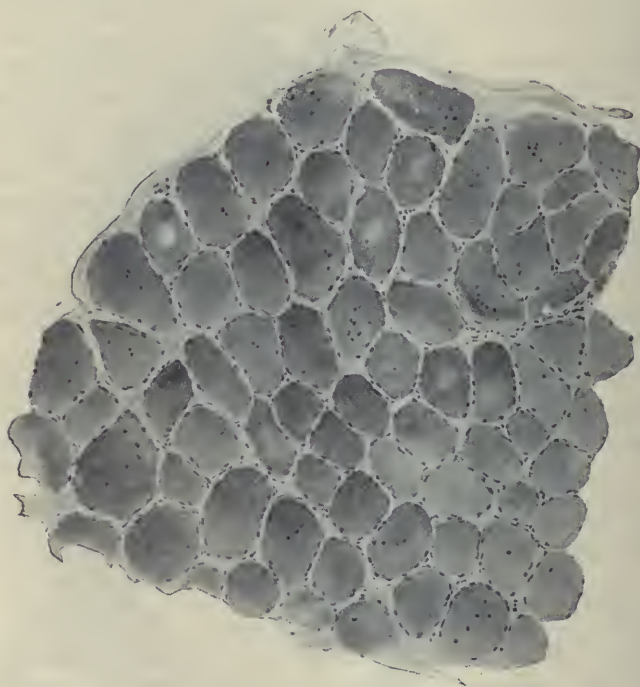


FIG. III.

Transverse section of non-extended muscle. (Oc. 4., Obj. A. Zeiss).

fibres were parallel, straight, with linear contours, while in the other there was no such regularity; the fibres not being straight and parallel, but convoluted. Furthermore, the transverse striation was here indistinct and delicate, the entire fibre being more homogeneous, and in some of the fibres only a longitudinal striation being discernable.

In short, our two sets of specimens show sections which in the one set closely approximate those from normal muscle,

while in the second they correspond fully to what has heretofore been looked upon as characteristic of Thomsen's disease.

Inasmuch as all the specimens were taken from the same individual, at the same time, and treated in precisely the same manner, this difference must be due to the accidental breaking away from its fastenings of the one piece of muscle; in other words, that piece of muscle which was allowed to contract to its limit showed the characteristic changes of Thomsen's disease, while the other not entirely contracted pieces gave sections which appeared almost like normal muscle. To this we shall refer again.

This case is as typical a case of Thomsen's disease as can well be found. The myotonic disorder, the myotonic reaction, the hypertrophy of the muscles, with their disproportionate weakness, all make the diagnosis unmistakable. Yet the absence of heredity, and especially the occurrence of the affection after an attack of typhoid fever at the age of 18, render it imperative to make use of some other designation than that of myotonia congenita.

The hypertrophy of the muscles coming on, as it did, at the same time as the myotonic disorder, and not having preceded it, shows the entire symptom complex to have been an acquired one. There is not the slightest ground for the assumption that any symptom of the disease existed prior to the attack of typhoid.

The result of the microscopical examination of the muscles is also of great interest, and it must cast a doubt upon the changes thus far described by myself and others as being pathognomonic of Thomsen's disease.

Another acquired case, but one which deviates very much from the congenital type, is the following.

I will curtail the history as much as possible.

D. L., male, 40 years of age, pianist by occupation, consulted me first about eight years ago. The family history is unimportant. Patient himself was perfectly well until his 26th year, when he met with an accident, slipping and falling. He at that time fell forward and struck flat upon his palms, over-extending both hands. This was followed by considerable

pain and some swelling in both wrist joints, but in about 10 days he had apparently entirely recovered, so that he again began to practice upon the piano, and, as was his custom, played from 6 to 10 hours daily. He then, after several days of such practicing, overstretched his right hand; this was immediately followed by a painless abductor cramp of the hand muscles, so that he could not bring his fingers together until he rubbed and warmed them, when the cramp passed away.

In the course of the following six months such cramps occurred whenever he forcibly stretched his fingers, i. e., abducted them, the left hand becoming similarly affected. He gave up piano playing, but without beneficial result, for the trouble grew constantly worse. Soon the cramps affected other muscular groups, finally involving all the muscles of the upper extremities, and occurring upon any forced movement.

The spasm is especially noticeable when he attempts to grasp any object tightly; then his hand clings to the grasped object, so that he cannot for the time being relinquish it.

He is worse in winter than in summer; cold water increases the liability to spasm, and he, therefore, always washes his hands in warm water. He has never complained of his legs troubling him.

Examination showed patient to be of strong and healthy appearance. With the exception of the muscular ones, no disorders of any kind are discoverable. The muscles of his arms, shoulders and neck are almost athletic in their development; the legs also are unusually large, but do not attract attention in the same measure as do his arms. The patellar tendon reflexes are exaggerated; the mechanical excitability of the muscles of both thighs is remarkably increased, a slight blow producing a quick contraction of the entire irritated muscle; tonicity is not present. Electrical reactions are normal.

The arms, in comparison to their development, are very weak; extension and flexion of the forearm upon the upper arm can very easily be prevented by comparatively slight opposition. A dynamometer, which under pressure by the average man indicates 150 for the left and 200 for the right hand, registers only 60 and 80, respectively, upon maximum pressure by the patient. Myotonic motor disorder is found to exist in all the muscles of the hands, arms and shoulders. In these same muscles, as well as the chest and neck muscles, the myotonic reaction to mechanical and electrical excitation was plainly demonstrable. The nerves (plexus brachialis, n. accessorius and n. ulnaris) show normal reactions to both forms of excitation. Rhythmical contraction waves were never obtainable

A few months ago I sought an opportunity of again examining this patient; he was then in a condition similar to that just described, but it was evident that the disease had made some progress. His entire upper body, with the exception of the abdominal and facial muscles, shows the myotonic disorders. So long as patient is careful to use his hands and arms only for ordinary movements, he has no trouble of any kind, but every sudden, violent exertion, or any occupation necessitating a quick maximum contraction of any of the above muscles or muscular groups, as well as sudden exposure to cold, brings on a spasm which is myotonic in character.

The myotonic reaction is present at all times.

His legs, with the exception of increased knee-jerks and hyperexcitability of the muscles to mechanical excitation, show no disorder.

Notwithstanding that this case differs materially from the typical picture of Thomsen's disease, in coming on after injury and overstrain in a man of 26, who had previously been in good health, and in affecting only the upper part of the body, and leaving the abdomen and legs entirely free, it must, nevertheless, be classed as a case of myotonia.

Whether or not it may be looked upon as a variety of Thomsen's disease is a question which is of no import, yet it is, aside from its acquisition, not any more atypical than the case of Martius and Hansemann, which Erb considers as such a variety, notwithstanding that the disorder occurred only temporarily under the influence of cold, was limited to the upper extremities, and showed no myotonic reaction during the free intervals.

It is to this class of cases that the term myotonia acquisita should be allotted, and, in my estimation, this term should be restricted in its use to cover only such cases. The term myotonia now describes a special form of spasm, characterized, as we have seen, by the myotonic motor disorder and the myotonic reaction; for all cases not showing at least these phenomena it would be better to make use of some other designation.

Thus Talma and Fürstner have published cases of

"myotonia acquisita" which certainly have nothing at all in common with the congenital form of the disease, as the name would seem to indicate. Talma's cases represent a series, showing an acquired tendency to spasm, occurring chiefly upon intended movements, the muscles involved, on account of their hyperexcitability, being easily thrown into a state of tonic spasm by various irritants. These spasms, however, increase in intensity the longer the muscles are used, while the reaction, which Talma describes as being similar to the myotonic reaction, shows upon careful interpretation nothing more than an abnormal hyperexcitability to mechanical and electrical irritants. In Fürstner's case, also, the myotonic reaction was not present, mechanical and electrical examination producing effects which were not totally dissimilar to this reaction.

It would, I believe, be better to designate all cases which show simply tonic spasm accompanying or following active movements, when unaccompanied by the other myotonic signs, by the term which Seligmüller has suggested, namely, *intention spasm* (a spasm occurring upon intended movements).

Such intention spasms have been described in connection with a variety of disorders, some having yet other symptoms in common with myotonia congenita, others admitting of no comparison at all with this disease. Such descriptions show that intention spasms may occur in disease of any part of the muscular and nervous system, and are especially often found in hysteria, tetany and occupation neuroses.

A case of this nature, but which showed so great similarity with the myotonic disorder and reaction that I am doubtful as to whether it is not, after all, a "myotonic condition," is the following: Perhaps it would be well to speak of such cases under the name of *myotonia transitoria*. This patient, L. G., was presented at the New York Neurological Society in May, 1892:

Cigarmaker; 34 years of age; Russian by birth. The family history, so far as obtainable, is negative, the father dying at 49 of some acute disease; the mother is living and healthy. The five brothers and sisters are healthy. Patient himself was always well, with the exception of a chancre in 1881. No secondary symptoms. He has been married five years; his wife has had three healthy children and no miscarriages.

About the end of February, 1892, spasms set in in the fingers of both hands; especially affected were the index and middle fingers. The spasm was limited to the flexor muscles of the fingers, and occurred only upon active movement, never spontaneously. Thus, so long as he kept his fingers open, there was no trouble, but as soon as he closed his fingers upon any object, a spasm ensued in the flexors, which prevented him from releasing the object grasped. The trouble gradually increased in extent, soon also involving the wrist.

He has never complained of pain. He comes for treatment because the trouble incapacitates him for work. He is able to do all the work which can be done with extended fingers, such as rolling the cigars on a flat surface, but when it is a question of finishing the point by turning with his fingers, he cannot let go of the cigar on account of the flexor spasm.

Examination, May 1st, 1892.—Muscular system not abnormally developed. The muscles of the hands, forearms and shoulders are, however, large and well formed, while the other muscles of the body are more flabby and smaller. The internal organs are normal. No fever. Urine contains neither sugar nor albumin. Pupils equal; react promptly. Ocular movements free; facial muscles unaffected. Tongue shows no deviation. Smell, taste, hearing, normal.

Upon intended movement the muscles of the hand, when these are closed forcibly, enter into a state of tonic spasm, so that the hand cannot be opened for a number of seconds. This intention spasm, in addition to the flexors of the hand and fingers, involves the adductors and abductors of the thumb and fingers, so that when the fingers are forcibly spread or voluntarily pressed together, they remain in either of these positions without being under control of the will.

These flexion spasms are most marked in the thumbs and 4th and 5th fingers, the 2d and 3d not being so much affected. The adductors and abductors of the thumbs and the deltoids show the following disorder:

Mechanical Excitation.—Quick, sharp blows by means of a percussion hammer produce marked contraction of the entire muscle (except in the deltoid, where only fibrillar contraction is produced). This contraction is slow and tonic in its formation and duration, lasting from 15 to 20 seconds. The same lasting contractions may be obtained by pressing or roll-

ing the muscles under any hard object. The mechanical excitation of the nerves is unchanged.

Electrical excitation with the faradic current showed, with strong currents, slowness and tonicity of the contraction, with an after-duration of 15 to 20 seconds.

With the galvanic current there was found in the same muscles an increased excitability, they reacting to very small currents, and tonicity, with after-duration, being produced by stronger currents (6 M. A.). The first tonic contractions were always obtained with the An. C., and it then required 1 to 2 M. A. more current to obtain a tonic Ka. C. C., while the relationship of the normal quick contractions obtained with minimal currents remained unchanged (Ka. C. C. > Aa. C. C.)

At that time no other muscles showed any changes whatsoever, functional, mechanical or electrical.

Three weeks later the biceps of each arm was found to present the same subjective and objective disorder as the other muscles. This condition lasted unchanged until September, 1892, when it gradually improved functionally, the My. R., however, persisting to the same extent.

In December patient claimed that he was perfectly well, and had been working at his trade uninterruptedly since two weeks. Examination failed to show any functional disturbances, and the muscles reacted normally to mechanical and electrical stimuli.

If we thus apply the term myotonia only to such cases as present the myotonic motor disorder and the myotonic reaction, and relegate all other spasms coming on upon voluntary movements to the category of intention spasms, our classification of myotonia congenita, myotonia acquisita and myotonia transitoria will at once make the class spoken of clear. The diagnosis of myotonia can present no difficulty, and its differentiation from tetany, pseudohypertrophy, spastic spinal paralysis, and even from Eulenburg's paramyotonia, need hardly be dwelt upon. In the latter affection not only are the specific myotonic reactions absent, but the muscular stiffness occurs only under the influence of cold, and not in consequence of muscular action.

The result of the microscopical examination of the muscle from Case 1 makes a reconsideration of the pathogeny of Thomsen's disease interesting.

Three theories regarding the nature of the disease, the psychopathic, the neuropathic and the myopathic, have each found adherents.

Against the psychopathic theory stands the entire weight of clinical evidence, which need not again be reviewed.

The myopathic theory, on the other hand, has had a great deal in its favor, especially the proofs deduced from anatomical findings. These are:

1. The hypertrophy and rounding off of the muscular fibres.
2. The increase of sarcolemma nuclei.
3. The diffuseness, indistinctness, and even loss of the transverse striation, and,
4. The minute changes which I have described as consisting in a massing together of the sarcous elements, and a correspondingly coarse appearance in some parts of the muscle, while in others these elements are minute, scarcely perceptible and widely separated from each other.

These anatomical findings are unfortunately all based upon the examination of pieces of muscle excised from the living body. The only autopsy recorded is the one described by Dejerine and Sottas,¹ which, while furnishing satisfactory proof to the reporters themselves of the purely myopathic nature of the disease, must impress every one else as being useless for purposes of argument.

Certainly many of the changes described in this report may be due to serous infiltration of the connective tissue and to secondary changes in the muscular parenchyma, thereby nutritionally produced. Furthermore, the cerebrum and cerebellum were not carefully examined, so that, even allowing that the muscular disintegration be not due to serous infiltration, its primary nature still re-

¹ This patient died of an acute nephritis with uræmic symptoms, and, aside from the muscular changes, the autopsy showed a marked serous infiltration of the entire body, hydrothorax, hydropericard and œdema of the lungs.

mains unproven. This autopsy, therefore, being unavailable as proof in support of the myopathic nature of the disease, we are again confined to that proof which may be derived from pieces of muscle.

The value of this proof has been seriously affected by the investigations of Oppenheim and Siemerling concerning the influence of excision upon pieces of muscle taken from the living body. Text books on histology, and different articles upon various topics, give the measurements of muscular fibres taken from the corpse as varying from 20 to 70 microns. The average fibre thus obtained measures 50 microns. Oppenheim and Siemerling found the following measurements in muscles excised during life:

1. Normal case; average, 69 microns; maximum, 106 microns.
2. Normal case; average, 93 microns; maximum, 121 microns.
3. Hysteria; average, 74 microns; maximum, 146 microns.
4. Hysteria; average, 69-93 microns; maximum, 140 microns.
5. Traumatic neurosis; average, 93 microns; maximum, 140 microns.

Inasmuch as the irritation due to excision causes considerable contraction of the excised pieces, as a result of which the primary fibres become shortened and correspondingly broadened, Oppenheim and Siemerling excised pieces of muscle from the adductor femoris of a living rabbit (a) simply; (b) after preliminary fixation upon a staff, so that they could not contract; (c) stretched upon a staff. The fibres of class a were all round, and measured from 46 to 99 microns in diameter; those of class b were all polygonal, and had a diameter of 22 to 66 microns, while those of class c measured from 9 to 33 microns. In addition to this preponderance of size and change in form of the simply excised fibres, they also revealed the presence of a greater number of nuclei.

The description of the extended and non-extended fibres from my case, therefore, conclusively proves that these observations may be directly applied to the human body, simply recognizing that my fibres were taken from a case in which the mechanical contractility of the muscles was very much augmented, and, therefore, the figures obtained must be higher than those obtained through similar excision of normal muscles.

Finally, we must draw the conclusion that the muscular changes hitherto considered pathognomonic of Thomsen's disease are merely the result of mechanical excitation of the muscles, and simply corroborate the clinical fact of their hypercontractility.

Admitting, however, the fact that the muscular changes found appear to be of a secondary origin, the existence of a functional disorder which allows the muscles to overact in consequence of excitation, cannot be denied. Whether this functional disorder lies primarily in the muscular or central nervous system is a question which cannot yet be answered.

Interesting, and, perhaps, casting some light upon the pathogenesis of this peculiar affection, is its occurrence in the case of W. G., after an attack of typhoid fever.

We know that many of the gross disorders of the nervous system are of post-infectious nature, thus it must be to-day acknowledged that multiple sclerosis is in very many instances the result of an antecedent acute infection.

That severe infectious diseases, as typhoid and diphtheria, through the toxins produced by their microbes, deleteriously influence the very impressionable nervous system is undoubted; and it has even been shown by Babès that the microbes themselves may migrate into the spinal cord and into the nerve cells, without causing any local lesion.

After the elimination of such toxins or microbes, the entire organism apparently again returns to its normal state, but who can say whether cells so acted upon are

not functionally altered. Certainly it has long been known that the nerve cells of adults who have passed through many sicknesses are not entirely normal, and the changes found have been fully described by Babès; changes which in healthy small children are never found. On the other hand, the nerve cells of adults often show no such changes. This fact can only be explained by the assumption of an inherent weakness in the nerve cells of certain individuals, while those of others possess more power of resistance.

There can be no objection to the statement that such an inherent weakness may occur hereditarily in many members of a family; just as whole families show an hereditary weakness of brain cells, and become neurasthenics. But this disposition in the majority of cases lies dormant until stirred up by some accidental cause. In the histories of muscular dystrophies the precedence of such an accidental cause in the shape of an attack of measles, scarlet, typhoid is not unusual, and it is not improbable that in the affection under consideration such a productive cause has in many instances also been at work.

It, therefore, seems to me permissible to look upon the disease as due to an embryonal developmental disorder of the nerve cells, consisting in the more or less diminished resistance of these cells to the influence of certain toxic processes, and that these intoxications then are in such predisposed individuals the direct producers of the disease.

Certainly, our knowledge of the pathogeny of this disease must remain obscure until a careful cytological examination of both the brain and spinal cord has been made.

LITERATURE.

- Talma S.: Deutsche Zeitschr. für Nervenheilkunde, p. 210, 1892.
 Fürstner: Archiv für Psychiatrie, vol. xxviii., p. 600, 1895.
 Erb, W.: Deutsches Archiv f. klin. Med., 1889, p. 529, vol. xlv.
 Susskand, A.: Zeitschr. f. klin. Med., vol. xxv., 1894, pp. 91-122.
 Jacoby, G. W.: Journal of Nervous and Mental Dis., March, 1897.
 Dejerine, J., and Sottas, J.: Révue de Med., xv., 3, p. 241, 1895.
 Oppenheim and Siemerling: Centralblatt f. d. med. Wissensch.,
 xxvii., 39, 41, 1889.
 Hochwarth, L. v. F.: Zeitschr. f. klin. Med., 1888, p. 424.
 Martius and Hanseemann: Virchow's Archiv, 1889, p. 587.

DISCUSSION.

Dr. Graeme M. Hammond presented three children, two girls and a boy, and all members of the same family, as examples of myotonia congenita. Two other children in the family showed no evidence of this disease, and were apparently normal in all respects. In the three cases shown the disease did not manifest itself until the eighth year of life; previous to that age, the children appeared to be like others, excepting that they were very dull, and made little progress at school; for example, the boy, who is now fourteen years of age, cannot spell words of even four letters. All three children had a peculiar facial expression; they looked depressed, and were subject to violent crying spells at the slightest provocation. One of the girls had had chorea, and subsequently developed the myotonic disorder. The family history was unimportant, and contained no other instances of this disease. All of the children had had chills and fever, and one of the girls still has these attacks occasionally.

The disease in the three cases was entirely confined to the hands and arms. The rigidity of the muscles was great, and the reflexes were sluggish. The electrical reactions were normal, except that tonic contractions were produced, the same as if the muscles had been struck. No muscular hypertrophy was noticed. The palms were well developed, but the strength of the arms and hands was much less than it should have been. The legs were not at all involved, but the knee-jerks in all three of the cases were almost entirely absent. The peculiar facial expression, Dr. Hammond said, was not due to rigidity of the facial muscles. No difficulty was experienced in moving the jaws, and no spasm of the ocular muscles existed. The hand could readily grasp an object, but relaxed with difficulty. The mental deficiency in these cases seemed to indicate some degenerative cerebral change, perhaps in the pyramidal cells of the cortex. Three cases occurring in one family also pointed to some congenital influence.

In conclusion, Dr. Hammond said he did not present these as typical cases of Thomsen's disease, but simply as cases of myotonia congenita.

Dr. Theodore Diller said he had had an opportunity to examine the first patient referred to in Dr. Jacoby's paper, and in that case the symptom complex was even more striking than in the three children shown by Dr. Hammond. In addition to the other symptoms, a distinct spasm of the extrinsic ocular muscles was produced by movement of the orbicularis palpebrarum. The patient could close his eyes tightly, but could open them only slowly, and with a distinct effort. All the reactions which Dr. Jacoby mentioned were

present. The wave of contraction, which various writers have referred to, could not be elicited. Dr. Diller said the case did not impress him as being of psychical origin.

Dr. J. J. Putnam inquired whether there was an abnormal shortening of the muscular belly, as occurs in muscular dystrophy.

Dr. Jacoby replied that such a change in the muscles had not been observed.

Dr. W. G. Spiller said he knew of two or three cases reported in the literature, in which intention spasm was present. Two were cases of syringomyelia, and the third, one of brain tumor. He had been permitted to examine the specimens in the only case of Thomsen's disease in which a necropsy had been obtained—that of Dejerine and Sottas. The lesions in the muscles resembled those described by Dr. Jacoby. The speaker said that from our present knowledge of the histological conditions, myotonia and progressive muscular dystrophy present histologically many features in common.

Erb has ventured the opinion that progressive muscular dystrophy may be due to functional changes in the cord. If this view is correct, the speaker thought it very remarkable that such a striking clinical picture as we see in advanced cases of this disease should result from changes which cannot be detected by the microscope. If Erb's theory regarding the spinal origin of progressive muscular dystrophy is correct, such a theory may be equally true of myotonia.

Dr. Jacoby, in closing, said that under the classification of myotonia only such cases should be included which present certain definite symptoms. In addition to the myotonic disorder and myotonic reaction, there should be an absence of anything pointing to gross disease in the central nervous system. Cases which do not fulfil these requirements should not be called myotonia, but should be classed under their proper designation, or designated as intention spasms. The so-called myotonic changes in the muscles, which have been described by various writers, and regarded as pathognomonic of this disease, have been found, by recent observation, to be not at all pathognomonic; the changes have been found to depend on the extensive shortening of the muscles, due to the method of preparing them for microscopic examination. In the case of Dejerine and Sottas, it was reported that nothing was found in the spinal cord, but the cerebrum and cerebellum were not examined. The change in the muscular tissues could be accounted for by the fact that these tissues were much infiltrated with serous fluid.

A CASE OF AMAUROTIC FAMILY IDIOCY WITH AUTOPSY.¹

By FREDERICK PETERSON, M. D.

This case was brought to my office in November, 1897. It was then an infant three months old, female, child of Russian Hebrew parents. The mother was 28 years old at the time of its birth, and had had five children and one miscarriage. Of the five children, the first was seven years old and normal, the second five and one-half years old (a blind idiot now on Randall's Island), the third a normal child of four years, the fourth a blind idiot, which died at the age of ten months, and the fifth is the case of amaurotic idiocy described in this paper. Thus there were three cases in this family. In the second child the blindness was not noted until the age of six months. In the fourth and fifth cases it was not noted until the infants were four weeks old. The blindness, however, may have existed at birth. My patient was sent to Randall's Island in January, 1898, and died there, March 16th, at the age of 7 months and 20 days.

The following history is from notes taken by myself and my internes at the Randall's Island Hospital for Idiots, Drs. Elizabeth Sturgis and F. O'Neil:

L. L., 7 months, 20 days.

Family History.—Mother, 28 years, German, healthy. Father, 29 years, Russian, said to have some lung trouble causing dyspnœa on exertion, but which is not consumption. History negative on both sides for syphilis, tuberculosis, insanity, epilepsy or nervous disease, as far as was known. No relationship exists between parents.

Personal History.—Born at term, labor normal; nursed four weeks and was then fed on milk and water, equal parts.

¹ Read before the American Neurological Association, May 27th, 1898.

Up to the time of admission, mother declared her to be as bright as her other children, laughing and playing with her hands, but crying much both night and day. Did not follow objects with her eyes, but pressed her hands into her eyes to a certain extent.

Examination showed a well-nourished baby, plump and of good muscular development.

Skin.—A *nævus* under chin and pigmented *nævus* on left thigh.

Eyes.—Media clear; pupils somewhat dilated, equally; a rotatory up and down movement, but no fine tremor; conjunctivæ, good color; owing to extreme restlessness of child, the fundus could not be seen. The eyes were examined by Dr. Percy Fridenberg.

Lungs, heart, liver and spleen normal.

Reflexes not increased, no rigidity, no paralysis.

Head.—Antero-posterior circumference, $15\frac{1}{4}$ inches.

Chest.—Circumference, $16\frac{1}{4}$ inches.

Length (of child).— $25\frac{1}{4}$ inches.

Child doing very well, but losing weight until Feb. 10th, then exposed to measles. Stools became green and watery; food was refused or vomited; temperature was raised, 101 to 102 deg. F.

Feb. 14th, coryza and distinct eruption seen on tonsils and throat.

Feb. 16th: Temperature, 104.

Feb. 17th: Sent to measles ward; no distinct rash ever seen on body. Gastrointestinal symptoms continued while in quarantine, refused food. About February 27th, rigidity of neck and knees, some twitchings of muscles. Heart and lungs negative.

March 9th, returned to Infant's Hospital. Became weaker, did not take much nourishment, and just before death she developed what was diagnosed as purpura.

Unfortunately the autopsy on this case was delayed fully forty hours, so that, although it was cold weather, and the body was well preserved, the examination of the finer nerve structures was to some extent interfered with. The autopsy was performed by Dr. D. Hunter McAlpin, Jr., to whose great courtesy I am indebted for the following notes, and also for the brain, spinal cord and parts of other organs used later for microscopical investigation.

Autopsy.—Body is that of a very emaciated child. Post-mortem discoloration present over abdomen and back. There is a large number of small hemorrhage spots of a purplish color in the skin covering the abdomen. There is a dark brown pigmented spot $\frac{3}{4}$ in. in length by $\frac{1}{2}$ in. in width on anterior surface lower third thigh, evidently a birth mark.

The skull-cap is of usual thickness.

The surface of the brain is markedly œdematous and is congested. The brain weighs 22 ounces. Placed at once in 4% solution of formalin for further examination. The pia mater of the spinal cord is congested. The cord is quite firm throughout.

The peritoneum is smooth and glistening. The intestines are distended with gas. The vermiform appendix is $1\frac{1}{4}$ inches in length, its lumen is patent. The mesenteric glands are not enlarged.

The pleura covering the upper and middle lobes is smooth; adherent to the costal and diaphragmatic surfaces of the pleura. The right lung is also adherent to the diaphragm.

The pleura over the anterior margin and in the interlobar fissure of the left lung is opaque. Apex of the lung shows a large emphysematous bleb. On section the upper lobe is of a pale pink color. On pressure a frothy straw colored fluid exudes. The pleura over the entire lower lobe is opaque and lustreless. Color of lower lobe is dark, mottled with few light areas. There are small nodules felt throughout the lower lobe. On section nodules are found firm, elevated and finely granular. On pressure a small amount of mucus exudes.

The pleura covering the upper and middle lobes is smooth; but over the lower lobe, posteriorly and inferiorly, it has lost its smooth and glistening appearance.

Upper lobe is light pink in color, and exudes a frothy fluid. At apex a mucopurulent material can be expressed from the bronchi. In the lower lobe there are few areas of consolidation similar to those in the left lung.

The pericardium is smooth and free from fluid.

The heart is of usual size. The right auricle and ventricle contain dark clots. Left auricle and ventricle are empty. Aortic valve normal. Endocardium pale and opaque. Heart muscle, firm. Color, pale pink. The other valves are normal. Right cavities dilated. Very small amount of subpericardial fat. The foramen ovale is closed by a membranous curtain, which is not adherent to the septum at its upper portion, so that a probe can be passed between the two auricles.

The kidneys are pale in color. The capsules strip off leaving smooth surfaces. The cortical portions have an opaque appearance. The markings are coarse.

The liver is normal in size. Borders shelving. Capsule smooth. On section liver tissue is dry. Lobules distinct. Other zones pale gray in color, blood vessel wall slightly thickened.

Spleen small. Capsule smooth. On section dark plum color. Glomeruli prominent. Consistency firm.

Left suprarenal, pale yellow color. Firm. Small cavity in centre. Right suprarenal, same.

Pancreas, negative.

Stomach, normal size. Contains a few milk curds and dark brownish material. Mucous membrane, thin, pale. Rugæ, obliterated. Intestines, distended with gas. Contain yellowish fluid material.

Two and a half feet above the ilio-cæcal valve is a Meckel's diverticulum measuring $\frac{3}{4}$ inches in length. It arises near the mesentery and has a small mesenteric attachment of its own.

Mucous membrane of small intestine apparently normal. Large intestine contains yellowish material. The solitary follicles are prominent.

Bladder, empty, mucous membrane normal.

Uterus, ovaries and Fallopian tubes appear normal.

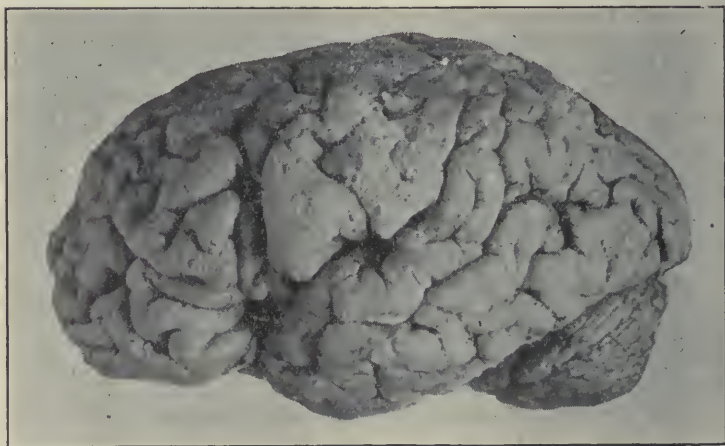


FIG. I.

Brain of amaurotic idiot. Exposure of insula; hypertrophic gyrus; confluence of central and Sylvian fissures.

Anatomical findings: Œdema and congestion of the brain, broncho-pneumonia, pleurisy, acute parenchymatous nephritis.

Macroscopic Examination of the Brain.—Grossly examined, this brain shows simply a few morphological features characteristic of defective development. On the lateral aspect (Fig. I.) we observe confluence of the central with the Sylvian fissure, exposure of the insula and one or two atrophic gyri. On the superior surface there is little to remark upon except unusual asymmetry (Fig. II.).

Microscopical Examination.—The microscopical investigation was carried out at the laboratory of the College of Physicians and Surgeons by Dr. James Ewing and the author, and the results are as follows:

The cortical areas about the calcarine fissures seem to be uniformly deficient in cells, and in some segments the number of cells appears very distinctly reduced. A striking feature of this portion of the cortex is the minute size of the cells, very few distinct somatochromes being seen except in the innermost layer. The separation of the cells into layers in this region is much less definite than is normal. The cells are moderately deficient in chromatic substance. There appears to be no difference in the structure of the right and left side in this region.

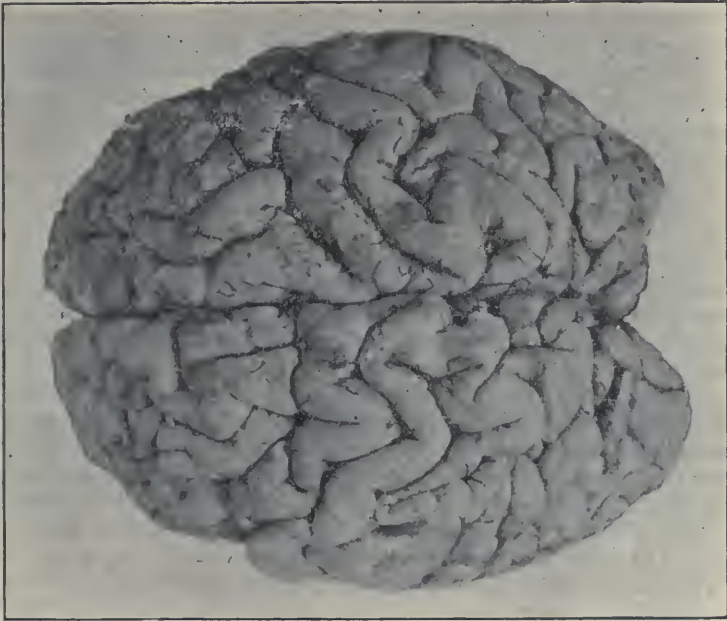


FIG. II.
Brain of amaurotic idiot.

In the motor areas the cells are markedly deficient in number, especially in the second and third layers, irregular in size, uneven in distribution, and uniformly deficient in chromatic substance. The abundance of large nuclei without demonstrable cell body in all layers of this and other regions suggests either deficient development or permanent atrophy.

In the hypertrophic lobule described in the left motor cortex the cells are markedly deficient in number.

None of the large stichochromes ordinarily seen in the motor cortex were anywhere found, but in some segments

rather large cells, forty to fifty microns in diameter were present, of nearly homogeneous appearance.

Throughout the frontal cortex, less distinctly in the temporo-sphenoidal lobe, the cells showed the same deficiency in number, and irregularity in shape and distribution, noted in the other regions.

Lumbar Cord.—Nearly all of the cells are quite normal. There are a few artificial changes, consisting in marked shrinkage of cell bodies and rupture of processes. Some cells show postmortem clouding of cell body and nucleus, and irregularity of chromatic masses. A very few cells show nearly complete absence of chromatic bodies.

Cervical Cord.—The anterior horn cells appear normal. A distinct group of medium-sized cells, situated in the posterior and external segment of the anterior horn shows typical axonal degeneration. Many posterior internal cells are moderately deficient in chromatic substance.

Nucleus XII.—The cells seem deficient in number, but show no distinct pathological alteration of the chromatic substance.

Nucleus X. (Superficial) and Nucleus Ambiguus.—The cells show moderate but uniform diminution in size of the chromatic bodies. In some cells the chromatic bodies are almost entirely absent, a few traces remaining at the periphery of the cell body only.

The limits of the third and fourth nuclei are indistinct. The cells of this region are deficient in number. Many appear shrunken. None of them contain well-formed chromatic bodies, but nucleus and cell body are homogeneous, diffusely stained, and contain few scattered granules of chromatic substance.

The olives are very well developed and cells are very abundant. The cells appear normal.

Purkinje's cells show only postmortem changes.

Corpora Quadrigemina.—The cells of this region are very deficient in number, usually of small size, ten to fifteen microns in diameter, belonging to Nissl's class of arkyochromes, although a few appear to lack distinct cell body, being classed therefore as karyochromes. A few cells also measure 30 to 35 microns in diameter. There are no distinct evidences of recent chromatolysis or of chronic atrophy. The tissue contains an unusually large number of arterioles and capillaries. In the deeper portion of the posterior corpus quadrigeminum are several groups of two to four large cells, 40 to 70 microns in diameter, circular on section, and closely resembling the cells of the posterior spinal ganglia. These cells contain an abundance of circularly arranged chromophilic bodies, and some of them show moderate central chromatolysis.

Internal Geniculate Bodies.—These nuclei contain a moder-

ate number of somatochrome cells, about 20 to 25 microns in diameter, and rather fewer karyochromes, both uniformly distributed throughout the tissue. The larger cells are arkyochrome in type and do not show any recent or old pathological changes.

External Genuiculate Bodies.—These bodies exhibit seven distinct layers of cells. There is first a narrow, superficial layer of small karyochromes with distinct cell body. The numbers of these cells vary considerably at different points. Beneath them, increasing in breadth, as one descends are six, dividing at times into nine, layers of larger arkyochromes, 30 to 40 microns in diameter, and among which are also a few karyochromes.

No pathological changes could be detected in any of these cells, other than deficiency in number. The arterioles and capillaries of this region are very numerous.

Sympathetic System.—In some of the sympathetic ganglia, lying on the aorta, near the coeliac axis, the cells are present in normal numbers and show various grades of central chromatolysis.

Nerve Trunks and Fibre Tracts.—For the study of fibres and tracts, sections were made of the occipital cortex of either side, the geniculate bodies, corpora quadrigemina, chiasm and optic nerves, at two points in the medulla, and in the cervical and lumbar cord. These sections were stained by Van Gieson's, Marchi's, and Pal's methods, but owing to the original fixative used, formalin, satisfactory results were obtained only from the first of these methods. In sections stained by picro-acid fuchsine, no distinct abnormalities were seen in any region of the central nervous system. The various tracts in the cord and medulla seemed to be normal in development, and the axis cylinders and myelin sheaths were intact. The optic nerves were not distinctly deficient in size or number of fibres. The optic chiasm and radiations were apparently normal.

It seemed probable, on comparison with sections of normal brains, that the development of fibres in the cerebral convolutions was moderately deficient, especially in the occipital region, but this condition was far less evident than the great deficiency of cells noted in these regions.

On comparison of the various sections of the cortex and cerebral ganglia in the present case with sections from the same region in normal infants, the abnormalities described became strikingly apparent. In the normal infant's brain the cells are arranged in very distinct vertical columns, as well as in longitudinal rows which in sections stained by methylene blue are plainly visible to the naked eye. In the present case nearly all traces of the vertical columns were missing, and the

separation into longitudinal rows was very indistinct. Moreover, the cortical cells were very deficient in number as well as in size and content of chromatic substance.

These changes were noted throughout the cerebral cortex, but were specially evident in the optic centres and ganglia. In the absence of demonstrable lesions in fibres and tracts, these cellular abnormalities, together with the increased number of blood vessels previously noted, constitute the main pathological features of the case.

Viscera.—Sections of the kidney, liver, spleen, lung, suprarenal and pancreas, failed to show any noteworthy lesions other than an advanced fatty infiltration of the liver.

Eyes.—The two eyes were removed and immediately placed in formalin. One was given to Dr. Carl Koller for examination, but his report has not yet been made. The other was given to Dr. Ward A. Holden, whose report is as follows:

One eye was received in formol 5 per cent. The retina was found to be detached at the macula, a postmortem change, which prevented the recognition of any existing gross pathological conditions. Sections cut in paraffin and stained by Nissl's method showed advanced postmortem changes: the vessels contained numbers of bacilli, the rods and cones were destroyed, the ganglion cells, bipolar cells, and nuclei of the rods and cones were vacuolated. In most of the ganglion cells the vacuolation had been so excessive that the cell bodies were more or less completely broken down so that nothing can be said as to their size in life. The nuclei took on a faint diffuse stain, and the nucleoli were well marked. Nissl granules were present. An attempt to stain the optic nerve by Weigert's method was not successful after the formol hardening. Hæmatoxylin-eosin preparations of the nerve revealed no pathological changes.

From this unsatisfactory examination of the eyes it cannot be said that pathological changes existed in life. If pathological changes in the ganglion cells existed, however, they were not in an advanced stage.

Conclusions: The brain shows, both macroscopically and microscopically, a condition of defective development, and this corroborates the findings in the several autopsies made in these cases (with the single exception of that of Hirsch, reported at this meeting). The pathological conditions are limited, as far as the fine structures are concerned, to the nerve cells of the cortex and medulla, which were found markedly deficient in number and in develop-

ment in the occipital region about the calcarine fissure, in the temporo-sphenoidal lobes, in the frontal lobes, in the motor areas, in the corpora quadrigemina and geniculate bodies, and in the third and fourth cranial nuclei. Postmortem changes did not affect the importance of these findings. As regards, however, the alteration of the chromatic substance of the cell, this must be referred largely to the general condition of the patient before death, and not to the disease under discussion. No definite changes in the fibres or imperfect developments of the tracts were discovered.

165. SUR LA VALEUR CLINIQUE DE PYRAMIDON (The Clinical Value of Pyramidon). R. Lépine (Lyon Médical, 85, 1897, p. 215).

This new claimant for antipyretic and analgesic honors is a derivative of antipyrine, and is said to be three times as effective and four times as poisonous as this drug.

The above author has employed it in twenty cases of various nervous ailments, and to the extent of his experience has nothing but praise for the new remedy. He has found it very efficacious in stilling pain, and has seen no ill effects of any kind from amounts up to 45 grains a day, the usual dose being four grains, three to five times daily. A case of tabes is instanced for the lightning pains of which the usual analgesics afforded no relief, even morphine in considerable doses being without effect, but in which 10 to 12 grains of pyramidon three or four times daily made the patient entirely comfortable for several weeks, at the end of which time the paroxysms of pain ceased. In all the other cases but one the drug relieved the pain from which the patients suffered, and this exception was an extremely bad case of neurasthenia with generalized pain that had resisted all other modes of treatment. In most of the patients the author compared the results obtained from pyramidon and 15 gr. doses of antipyrine, and without exception the former remedy was preferred.

He has also used the drug as an antipyretic in typhoid fever with excellent results, but considers that his experience is as yet too limited to draw positive conclusions. He feels sure, however, that the remedy is of real value and merits an extended trial.

PATRICK.

THE PATHOLOGICAL ANATOMY OF "A FATAL DISEASE OF INFANCY, WITH SYMMETRICAL CHANGES IN THE REGION OF THE YELLOW SPOT" (WARREN TAY), "AMAUROTIC FAMILY IDIOCY" (SACHS), "INFANTILE CEREBRAL DEGENERATION" (KINGDON AND RUSSELL).¹

By WILLIAM HIRSCH, M. D.

The first case of this peculiar disease has been described in the year 1881 by Warren Tay, under the name of "Symmetrical Changes in the Region of the Yellow Spot in Each Eye of an Infant." Since that time a number of other cases have been brought to light by different observers, so that the total list up to to-day amounts to 26 cases, including my own. For quite a number of years after Tay's first publication, various observers paid almost exclusive attention to one particular symptom of the disease—that is, to the peculiar ophthalmoscopic condition. Only after some time was it realized that we had to deal, not with a localized affection of the eye, but with a more extensive disease of the central nervous system. The credit for having first called the neurologists' attention to this peculiar affection belongs to B. Sachs. The clinical symptoms have ever since been so accurately described that nothing new can be added in this respect for the present. The main features of the disease, which are almost absolutely uniform in all cases, are the following:

The parents of these children are strong and healthy, and give no history of syphilis or tuberculosis. They all belong to the same race; they are, with few exceptions,

¹ The figures accompanying this paper are photographs from diagrams made for a demonstration at the twenty-fourth annual meeting of the American Neurological Association, on which occasion this paper was read.

eastern Jews. There are always several children of the same mother affected. In some cases there are healthy children between the affected ones; in others, all the children in the family are affected in the same way. The sex seems not to have any influence on the disease. The children are born apparently in good health and develop normally up to the third or fifth month of age. Between the third and eighth month the muscles begin to become flabby and weak. The child is no longer able to sit up or to hold up its head. The reflexes remain present, as a rule. In some cases the extremities become rigid and contracted; mental development is arrested; the children become dull and apathetic; the eyesight gradually diminishes to complete blindness. In most cases there is a marked hyperacuity, and some children are hypersensitive to touch. The most characteristic symptom, which, in fact, gave rise to the discovery of the disease, is the peculiar changes on the retina. The assumption of some observers, that these changes are congenital, is apparently erroneous. When the peculiar ophthalmoscopic picture has developed, we find in the region of the yellow spot a whitish opacity, the centre of which shows a cherry-red spot. The discs appear at first normal, but undergo atrophy later on. The following is the history of my own case:

L. P., son of healthy parents of the eastern Jewish race, was brought to my clinic in July, 1896. The child, which was then ten months old, had developed well until the age of six months, when it began to be weak in the back, so that it lost the ability to sit up. The mother stated that she had lost two other children at the age of eighteen and twenty months, respectively, which apparently presented the same condition as the patient. The muscles of the whole body were very flabby, and the child was unable to hold up its head or to sit without support. It had normal perception of light, and there was marked hyperacuity—a symptom which the mother had also observed in the two other children. The reflexes were present; sensation was normal; the internal organs were also in a normal condition. The ophthalmoscopic examination

revealed the typical picture described above. The child continued to become gradually weaker, until it died, on the 27th of July, 1897, twenty-two months old. An autopsy was made four hours after death.

So far five autopsies have been performed—two by Sachs and three by Kingdon and Russell. In the first case of Sachs, only the cortex of the brain was examined; in the second case, sections were made through all parts of the cortex, of the ganglia, of the optic chiasm, of the pons, the medulla and the cervical cord. The result of the examination of these two cases was alike. There was an equal change of the pyramidal cells throughout the whole cortex of the brain. The contours of the cells were rounded, and the cells exhibited every possible change of their protoplasmic substance. In the neuroglia there were no evident changes, nor was there any distinct sclerosis in any part of the brain. The blood vessels were found perfectly normal in every respect. The spinal cord, it was said, contained no changes in the gray matter, but exhibited a degeneration of the lateral areas, including the pyramidal tracts, and extending to the periphery. From these findings the conclusion was drawn that the disease was confined to the cells of the cortex of the brain, and consisted of an arrested development of this organ.

• Quite in accordance with this view was the result of the three autopsies of Kingdon and Russell. They found the same changes in the pyramidal cells of the cortex, and state expressively that the basal ganglia, the cerebellum, the cells of the posterior column nuclei, and the gray matter of the spinal cord exhibited no changes whatsoever. In the lateral tracts they found the same degeneration which had been described by Sachs.

On the strength of these findings these authors also were led to believe that the disease was limited to the cortex of the cerebrum. They did not, however, agree with Sachs in the assumption of an arrested development, but considered the condition a degenerative process. The nature of this process was left an open question.

In accordance with their respective views, Sachs published his first cases under the title of "Arrested Cerebral Development" and "Agenesis Corticalis," while Kingdon and Russell called the disease "Infantile Cerebral Degeneration." Recognizing the insufficient anatomical base on which the disease stood at that time, Sachs preferred a clinical designation, and proposed the name of "Amaurotic Family Idiocy."

I will now refer to the result of the post-mortem examination of my own case, which I hope will throw some light on the different questions:

As stated before, the autopsy was made four hours after death. The skull was thick and symmetrical, and both fontanelles were closed. The dura was adherent to the skull. The pia could easily be removed from the brain. The macroscopical inspection of the brain showed no abnormal conditions. There was no œdema of the convexity, and no increase of fluid in the lateral ventricles. There were no abnormalities of fissuration, except an unusual prolongation of the second temporal fissure. The entire brain, with the medulla oblongata and the spinal cord, was put immediately after the removal in a 10 per cent. formaline solution. Both the eyes were removed, and one put in formaline and the other in Müller's fluid. After the hardening process the whole central nervous system was cut into small pieces and imbedded in paraffine. Serial sections, varying in thickness from one to four microns, were made through the whole spinal cord, the medulla oblongata, corpora quadrigemina, optic thalamus, nucleus caudatus, chiasm, optic nerves, and through the entire cortex of the cerebrum and cerebellum. The sections were stained with various aniline dyes, as methylene blue, eosin, thionin, nigrosin, fuchsin and with different kinds of hæmatoxylin.

For practical reasons I will begin the description of the different sections with that of the spinal cord. I found the same degeneration of the pyramidal tracts which had been described by the other observers. But, in addition to this, there are very pronounced changes throughout the entire gray matter of the cord. All the nerve cells of the anterior, as well as of the posterior, horns are enormously enlarged. They appear blown up and round in shape. The nucleus is well defined, and invariably moved toward the periphery of the cell body. It contains a nucleolus which is stained dark and

has a sharp outline. The cell body is surrounded by a membrane. On account of the enormous enlargement, the cells appear much more numerous than normally, so that some parts of the gray matter seem to consist almost exclusively of cells. The most peculiar aspect is offered by the motor cells of the anterior horns. (Fig. I.) They are more than twice their normal size. Many of them appear as round masses, without any nucleus. This, however, can be shown by serial sections to be due to the direction of the cut through the cell. In fact, all the motor cells contain a nucleus and nucleolus. Some of the cell bodies are vacuolated, the vacuoles being lined with a membrane. The processes of the cells, the dendrites, as well as the axis cylinder, seem to be very few, and often they appear to be broken off the cell. In most in-

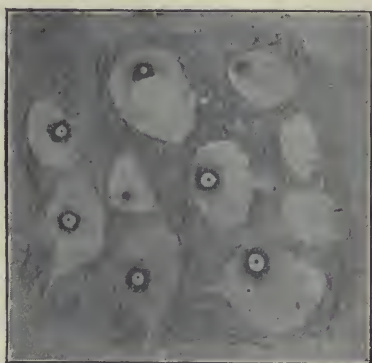


FIG. I.

Cross-section through the anterior horn of the cervical part of the spinal cord.

stances the cells are immediately surrounded by connective tissue, the pericellular space being small or missing altogether. The nucleus, which is invariably situated near the periphery of the cell body, is surrounded by a dark zone, which gradually shades off into a more or less colorless area. When we come to study the minute structure of these cells with a high power, we find that the Nissl bodies have disappeared entirely. The dark zone around the nucleus consists of a granular mass, which is probably formed by broken-up Nissl bodies, which appear to be in a condition of pulverization. The light area of the cell body is made up of a very fine network. In a very few instances the dark zone around the nucleus contains a few normal chromatic bodies.

The cells of the posterior horns (Fig. II.) are also changed in the same manner. They, too, through their enormous en-

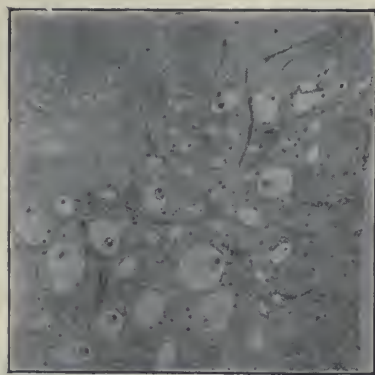


FIG. II.

Cross-section through the posterior horn of the cervical part of the spinal cord.

largement, appear very numerous, and wherever the section has occurred, above or below the nucleus, the cell looks like a big, round mass, without any definite structure. As we approach the medulla oblongata, we find the same changes in

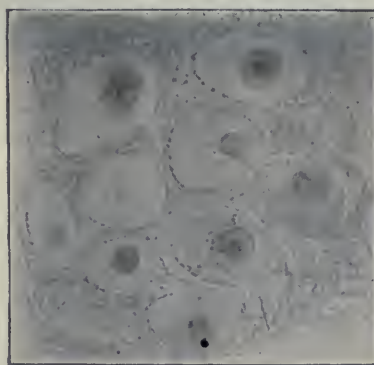


FIG. III.

Cross-section through the motor nucleus of the trigeminal nerve.

all the nerve cells; not only all the nuclei of the cranial nerves, from the hypoglossus to the oculomotorius, but also the cells of the reticulum, the olivary bodies, the pons, are all changed

in the same typical manner. According to the conditions of their natural shape, they appear also different in their morbid condition. The cells of the olivary bodies are round in shape, showing a sharply defined nucleus and nucleolus. The cells of the reticulum and the pons appear as oval-shaped bodies, and, owing to the fact that the nucleus is pushed to the periphery of the cell body, a great many cells are cut so that the nucleus does not appear in the section.

There are some groups of cells which seem to have undergone still further changes of decomposition. Such groups are principally found among the nuclei of the cranial nerves. The motor nucleus of the trigeminal nerve, for instance, contains cells which exhibit this condition. (Fig. III.) The nucleus has lost its sharp outline, and the nucleolus can hardly be



FIG. IV.

Cross-section through the nucleus ambiguus.

recognized at all. The cell body is filled by irregular masses—apparently detritus of the normal protoplasm. Such masses of detritus are especially accumulated at the periphery of the cell body, and also in the pericellular lymph space. Cells of other groups, as, for instance, in the nucleus ambiguus, are remarkable for their peculiar elongated shape. (Fig. IV.) The dark zone around the nucleus shows distinct traces of the Nissl bodies, and the rest of the cell body, as in the cells of the anterior horns, is made up of a fine network. The nucleus appears as a homogeneous body, and the darkly stained nucleolus contains a light spot in its centre. The cells of the sub-cortical ganglia, the optic thalamus, the nucleus caudatus and the corpora quadrigemina exhibit exactly the same condition as the other cells. The cells of the substantia nigra show the

pigment very plainly, and otherwise exhibit the same changes of the nucleus and cell body.

The cortex itself, which was examined throughout the entire brain, shows the same typical changes all over. (Fig. V.) The different layers of the pyramidal cells can still be well differentiated; the cells themselves, however, are changed in the same typical manner. They are all considerably swollen, so that at some regions hardly anything but cells is to be seen. The nucleus is displaced to the periphery of the cell body, and in some instances projects considerably beyond the surface. The cells are nearly all of oval shape, and there is not a single cell in all the specimens which has retained its pyramidal character.

Comparatively small changes are found in the cerebellum.

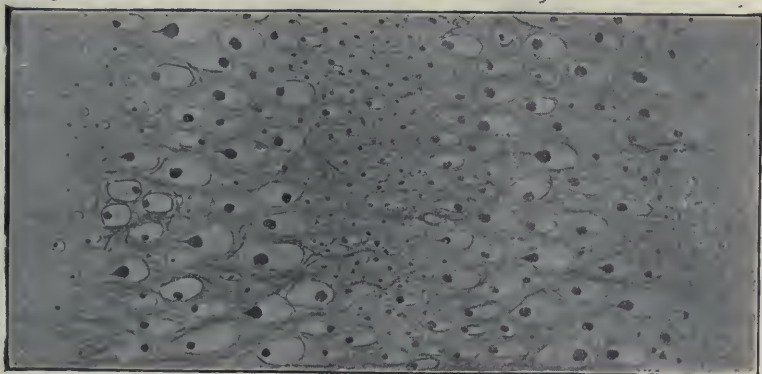


FIG. V.

Cross section through the cortex of the first frontal convolution.

The granular area shows no abnormal condition at all, while the Purkinje cells appear to be affected in some way, showing a much less distinct structure than under normal conditions, without, however, exhibiting the enlargement of the circumference of the cell body that was observed in all the other nerve cells of the nervous system.

The cross sections through the optic tract and the chiasm showed complete degeneration. The cauda equina was found to be perfectly normal, and so were all the roots of the spinal cord.

The eyes have been kindly examined by Dr. Holden, whose detailed report follows this paper.

The ganglion cells of the vesicular layer of the retina have been found in the same swollen condition as the cells of the

central nervous system, and it is due to this condition of the cells that we find the peculiar ophthalmoscopic picture in this disease. As is well known, the only part of the retina in which the vesicular layer consists of more than one single layer of ganglion cells is the macula lutea. Here we have an accumulation of from six to seven layers of cells, and it is easily understood that when these cells become enormously enlarged, and their bodies offer an opaque character during life, they will produce the peculiar whitish opacity in the region of the macula.

So, then, we have an equal affection of all the nerve cells of the entire nervous system, the main features of which are a condition of chromatolysis and other degenerative processes of the protoplasm, combined with considerable swelling of the cell body and displacement of the nucleus toward the periphery of the cell. The neuroglia has been found to be perfectly normal, and no affection of the blood vessels could be noticed through the entire system.

There seems to be a discrepancy between these findings and those of the previous observers, the latter claiming only an affection of the cerebral cortex. This, however, seems to be due to the differences of the hardening process. Sachs, as well as Kingdon and Russell, has hardened the brain and cord in Müller's fluid, which does not permit of the study of the minute structure of the cells. Especially, changes in the smaller cells, as those of the posterior horns of the cord and the medulla oblongata, cannot be recognized in specimens hardened in Müller's fluid. That, in fact, Sachs' second case—in his first case only the cortex had been examined—offered the same change of the motor cells in the anterior horns of the cord as in my case, can easily be recognized from his specimens even to-day. Dr. Sachs was kind enough to put some of his specimens at my disposal, and when we came to compare them with my own, it was evident that we had to deal with perfectly analogous conditions. Although the Müller's fluid specimens do not show the pe-

culiar structure of the cells, they show, however, sufficiently well the enlargement of the circumference of their bodies in the anterior and posterior horns, which could easily be overlooked, since changes in the structure of the cells necessarily escape observation. Inasmuch as the positive findings of Kingdon and Russell, whose cases were also hardened in Müller's fluid, are in perfect harmony with those of Sachs and myself, I do not hesitate to assume that their negative statement also could be modified in the same way as Sachs', and that the morbid changes in their cases, too, were not confined merely to the cortex of the brain.

So, it seems, then, to be an established fact that we have to deal not, as it was supposed, with an isolated cortical lesion, but with an affection of the entire nervous system. The next question to be decided would be: Of what nature is this affection? Have we to deal, as Sachs claims, with an arrested development, or, as Kingdon and Russell maintain, with a degenerative process—that is, an acquired disease?

Against the theory of an arrest of development stand both the clinical and anatomical facts. It is generally stated that these children are born in good health and well developed. In my own case the intelligent mother, who had previously lost two children of the same disease, watched the baby with the greatest anxiety, and still could not detect any morbid sign before the age of six months. Of great importance for the proper judgment of the nature of the disease is the fact that the peculiar ophthalmoscopical picture develops only comparatively late, sometimes long after the other symptoms have made their appearance. Since we know now that the peculiar condition of the macula lutea is due to a certain change of the ganglion cell layer, we are justified in assuming that the cells have been normal before the appearance of this peculiar picture. Besides this, there is no analogue in the whole embryology to this special affection of the

nerve cells, nor could any arrest of development account for an affection of all the nerve cells, and nothing but the nerve cells, of the entire system.

If, then, we have to deal with an acquired disease, of what nature and of what origin could such disease be? The total absence of any affection of the blood vessels, as it could be stated in my case, as well as in the other cases, justifies us in excluding any inflammatory process. Primary idiopathic diseases of the nerve cells occur only as systemic diseases; but no disease is known in which simultaneously all the nerve cells of the entire system become diseased. Besides, in systemic diseases the anatomical picture of the cells is quite different from what we have found here. There we have the typical picture of atrophy of the cell, while here we have an anatomical condition which does not correspond to any of the idiopathic cell affections at all.

The only theory, then, which is left, and which in fact corresponds in every respect to the clinical and anatomical aspect of these cases, is to assume a toxic condition; to assume the action of a poison on the nerve cells. That poisons are really apt to act that way is a well-established fact. The anatomical picture of these cells also corresponds in every respect to those found after experimental poisoning. The chromatolysis, the swelling of the body, the displacement of the nucleus, the destruction and breaking off of the dendrites and the axis cylinder; all these are well known features of poisoning, and, in point of fact, symptoms unknown in any other condition. We have to distinguish between two different ways in which a poison might affect the nerve cells. It might either act directly on the cells, or it might produce a disturbance in the nutrition, and so, by changing their metabolism, cause a degeneration of the protoplasm. The total absence of any morbid changes in the blood vessels or in the neuroglia, the fact that the pathological changes are confined to the nerve cells exclusively, lead me to believe that the

virus, whatever it may be, exerts a direct action on these cells, an assumption which I think finds further support in the anatomical appearance of the cells themselves.

The next question, then, would be: Of what nature is this poison, and where does it come from? This question cannot be answered yet with any amount of certainty. It would, of course, seem very tempting to assume a toxic condition of the mother's milk, as this would also easily explain the family type of the disease. But there are other possibilities to be considered, and so this question must be left open for further investigation. So much, however, I think can be said with certainty to-day—that this peculiar affection does not consist of an arrest of development, but is an acquired disease affecting the nerve cells of the entire system, produced by some poison. I would suggest that as soon as the diagnosis of such a case has been made, the child be taken from its mother's breast, and all future children be fed with other nourishment. Future investigations will have to be directed toward the clinical examination of the blood, and the excretions of these children, and of the milk of the mother, and it may be hoped that by these means all questions as to the nature and etiology of this interesting disease may be definitely determined.

-
166. KYSTES DU CERVEAU (Cerebral Cysts.) Jaboulay, (Lyon Médical, vol. 87, Jan. 23d, 1898).

This is a brief report of operation on eight non-parasitic cysts of the brain by an author who is very active in cerebral surgery. Most of the patients were epileptic, and the majority of the cysts were secondary formations following cranial injury. By far the best result obtained was in the case of a young man who, a year before, had sustained a fracture of the skull, for which a trepanation was done. He developed epilepsy, and in the cicatricial tissue a cyst was found and emptied. It is noted that after the operation "the seizures disappeared almost completely." Of the remaining seven cases, the patients either succumbed to the operation, were entirely unrelieved or showed a "slight amelioration," with the exception of the last, who had been operated upon only a month before, and who seemed decidedly improved. On the whole, the report shows again the fruitless results of indiscriminate operations on the brain. PATRICK.

PATHOLOGICAL REPORT ON THE EYES OF DR. HIRSCH'S PATIENT WITH AMAUROTIC FAMILY IDIOCY.¹

BY WARD A. HOLDEN, A. M., M. D.,
New York

The upper diagram in Chart A represents roughly the ophthalmoscopic picture characteristic of amaurotic family idiocy, and the lower diagram represents a corresponding section of the posterior part of the eyeball passing through the optic disc and macula lutea. In these cases the macular region is occupied by an oval, gray patch about twice as broad as the optic disc, and having a red spot in its centre. In nearly 30 cases that have been reported the eye changes have been remarkably uniform.

As regards the explanation of this fundus picture, which has often been compared to the change following embolism of the central artery of the retina, clinicians have been inclined to believe it to be due to a retinal oedema, rendering the retina opaque, and obscuring the normal red color of the choroid, except in the fovea centralis, where the retina is very thin. This view was taken also by Treacher Collins, the former pathologist of Moorfields, who made a microscopic examination of the eyes of two patients. He reported that there was a spacing out of the tissues in the outer reticular layer, indicative of oedema, and no apparent changes in the other layers. But he regarded his examinations as unsatisfactory, since in all the eyes examined the retina about the macula was thrown up into a fold, a common postmortem change. The retinal elements in such folds are always spaced out, so that a deceptive appearance of oedema is presented even in healthy eyes. The fact that Collins noticed no changes in the ganglion cells is not surprising, since little atten-

¹Read at the twenty-fourth annual meeting of the American Neurological Association, May, 1898.

tion is given to the retinal ganglion cells, as a rule, and scarcely any attempts have been made to study them by modern cytological methods.

The eyes on which I wish to report were removed by Dr. Hirsch four hours after death, and were hardened, one in Müller's fluid and one in formol, 10 per cent. In the formol eye the retina remained in position at the macula, and, after hardening, the gray patch at the macula was

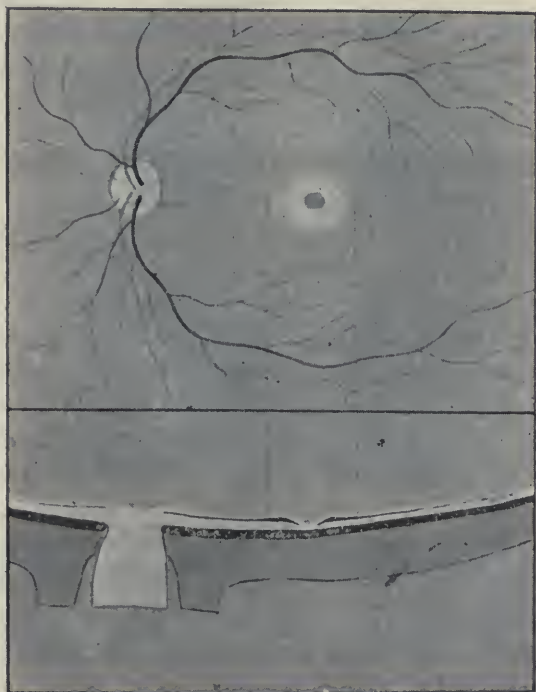


CHART A.

Upper diagram representing the fundus picture; lower diagram representing a corresponding section of the eyeball; the dark line in the retina indicating the layer of ganglion cells.

seen as in life. Horizontal and vertical sections were cut through the maculas, and longitudinal and transverse sections through the nerves, after celloidin and paraffin imbedding, and every applicable stain was used. The findings were checked in every possible way by comparison with other eyes, in order to rule out errors of interpreta-

tion. The eyes of an infant dead of the same disease, pneumonia, were prepared in exactly the same way, and cellular alterations due to postmortem changes, strong hardening solutions, and various general diseases were studied at great length. The work was done in the Pathological Institute of the New York State Hospitals.

In the normal retina the ganglion cells vary from 10 to 30 microns in diameter, and in the periphery of the retina where the cells are scattered, large cells abound, but in the macular region, where the ganglion cell layer is several

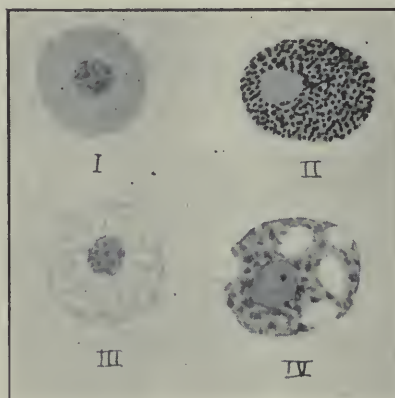


CHART B.

Fig. I. A retinal ganglion cell stained with hæmatoxylin-eosin. Fig. II. The same with Weigert's hæmatoxylin. Fig. III. The same stained by Nissl's method. Fig. IV. A retinal ganglion cell from Dr. Peterson's case stained by Nissl's method.

cells deep, the cells are mostly small, irregularly pear-shaped, and packed close together.

Sections of the Müller's fluid eye stained with hæmatoxylin-eosin showed the average size of the ganglion cells to be increased and the average shape to be altered toward the globular. (Fig. I., Chart B.) The nuclei were mostly near the centre of the cells. The cell body with this stain appeared homogeneous, but when similar sections were stained by Weigert's method or with hematein the entire body of the cells appeared to be filled with coarse, black granules. (See Fig. II.)

In Nissl preparations of the formol eye, at first glance, nothing was seen of the ganglion cells but their nuclei, which stained deeply and were coarsely granular. (Fig. III.) Under a 1-18 immersion the cell membrane and the cyto-reticulum could be clearly made out, but no Nissl granules were present, and the cell body had the appearance of having had its liquid contents withdrawn, leaving the naked cell framework. (Fig. III.) These staining reactions characterize the cell absolutely.

Furthermore, comparison with Dr. van Gieson's hematein preparations of the brain of Dr. Sachs' patient, which was hardened in Müller's fluid, and comparison with Dr. Hirsch's Nissl preparations of the brain of his patient, hardened in formol, showed that the staining reactions in the cerebral and the retinal cells in each case, allowing for their difference in size, were identical. In the eyes of Dr. Peterson's patient, who died at seven months of age, the autopsy being made forty hours after death, postmortem vacuolation was found in the ganglion cells (Fig. IV.), but the nuclei were not granular, and the nucleoli were distinct, while Nissl granules were present in the cell bodies; the advanced changes found in both nuclei and cell bodies in Dr. Hirsch's patient were here wanting.

No actual œdema of the retina was found on careful comparisons with preparations of normal eyes cut in the same directions and thicknesses. The other layers of the retina were apparently normal, excepting the rods and cones, which in their outer segments had undergone the usual early postmortem change. The ciliary nerves were normal, and no ganglion cells giving these peculiar staining reactions were found in the ciliary body.

The optic nerve showed an absence of the myelin in many fibres in each bundle, and the neuroglia tissue was increased—in other words, the condition was that of simple degeneration.

The changes in the ganglion cells readily explain the

fundus picture, which, indeed, admits no other anatomical explanation.

In the fovea centralis ganglion cells are practically absent. At the margins of the fovea the ganglion-cell layer abruptly increases in thickness, until it is from 6 to 10 cells deep. A disc diameter from the fovea, horizontally in either direction, the ganglion-cell layer has thinned down to a layer from 3 to 4 cells deep; two disc diameters from the fovea it is from 2 to 3 cells deep, and beyond this it thins down further to a layer from 1 to 2 cells deep, while in the periphery of the retina it is a single broken layer of scattered cells (see diagram, Chart A). Above and below the fovea centralis, in the vertical meridian, the ganglion-cell layer thins out rather more rapidly than in the horizontal meridian. The oval gray patch occupies, therefore, exactly that portion of the retina in which the layer of enlarged and altered ganglion cells is four or more cells deep. At the margins of the patch, where the ganglion-cell layer is thinner, the faint gray color fades away gradually into the normal red of the fundus. Near the fovea centralis, where the ganglion-cell layer is thickest, the gray color is most intense, and in the fovea centralis, where ganglion cells are wanting, the red color of the choroid shows through, appearing darker by reason of contrast with the surrounding light gray.

Tay, who first described this disease, and also Kingdon have followed the course of the fundus changes. They have found that the fundus is normal until after the third month. Then a haziness appears in the macular region, and the usual picture is fully developed in the fifth or sixth month. At this time the infant often has enough vision to follow a light. A month or two later, the macular picture remaining the same, vision is lost, and the optic nerve becomes atrophic. Although the macula lutea is not fully differentiated at birth, its layer of ganglion cells is five or six cells deep, and the fact that the retina is then ophthalmoscopically transparent indicates that the gan-

glion cells cannot be much affected, if at all. The optic nerve atrophy which follows the appearance of the macular changes is to be interpreted, perhaps, as both an ascending and a descending degeneration—a breaking down of the neuraxons of the affected retinal ganglion cells and of the affected ganglion cells in the basal ganglia.

We have thus found a common explanation for the symptoms of this complex affection, and have shown it to be pathologically a unit. The ocular neurons have undergone a primary alteration, like the cerebral neurons. Nor should this cause surprise, when we remember that the retina and the optic nerve are genetically a portion of the brain, being developed embryologically from the same medullary tube of involuted ectoderm. Embryologically related to the brain, the inner layers of the developed retina are so similar in histological structure to the cerebral cortex that they are known as the cerebral stratum of the retina. Analogous, embryologically and histologically, we have seen also that the retina and cortex may suffer analogous primary pathological changes. And in future investigations of nervous diseases, we must consider the retina a division of the central nervous system, which is quite as worthy of study by the pathologist as are the brain and cord.

DISCUSSION.

Dr. B. Sachs said that it was a great satisfaction to him that a paper read by him on this subject before the association, eleven years ago, had led to so interesting a discussion. He was firmly convinced that amaurotic family idiocy was a morbid entity, which he knew would be placed upon a firmer basis by the extensive pathological studies made by Drs. Hirsch, Holden and Peterson. The speaker said that when he first entered upon the study of this special disease, he had no theory in mind as regarded its causation, and even to this day he had not formulated any theory, nor did he think that the time for such a formulation had arrived.

Dr. Sachs stated that considerable emphasis had been placed upon the circumstance that a disease which was supposed to be a congenital one did not apparently develop its

symptoms until four or five months after birth. His answer to that statement was that the latter fact did not prove that the disease was not congenital, because it was extremely difficult to prove that a child under the age mentioned was perfectly normal in every respect. Moreover, in a case which had been under his observation, he had been able to satisfy himself that the child, when only three months old, was mentally deficient. The statement of the mother in regard to the mental condition of the child at a very early age was not always conclusive. As these patients grow older, both the mental and physical deterioration become more marked, and many of them die in a condition of marasmus. This last fact he wished especially to emphasize.

As regards the seat of the lesions in these cases, the speaker said that Dr. Hirsch had done excellent work in showing that the disease is not restricted merely to the brain or cerebral cortex, but may affect the entire nervous system, including even the cells in the gray matter of the cord. The cellular changes are similar throughout the central nervous system, and the pathological findings of Dr. Hirsch were entirely in accord with those in two cases of his own, one examined eleven and the other nine years ago. The speaker said that in his earlier researches on this subject he had interpreted the disease as being chiefly cortical in origin, but even at that time he had a suspicion that the lesions were not confined to the cortex, and Dr. H. Knapp, who had examined the eyes in his first case, insisted at the time that it would some day be found that the changes in the optic nerves were parallel to the changes in the cortex. In Dr. Sachs' second case the changes were present in the brain and spinal cord; the changes in the latter region he at first ascribed to secondary degeneration, but later on he came to the conclusion that they were the result of arrested development. Kingdon and Russell, on the other hand, were the first to claim that the changes were due to degeneration. Dr. Sachs was of the opinion that an arrest of development is necessarily followed by a degeneration, and for that reason he did not think there was such a radical difference between the interpretation put upon the cases by Kingdon and Russell, and now by Dr. Hirsch, and that of himself.

With regard to the cause of this degenerative process, Dr. Sachs said that, while the theory advanced by Dr. Hirsch was very interesting, it was probably influenced to some extent by the theory of another illustrious author, who had attempted to explain hereditary influence on the basis of a toxic infection. The speaker said he could not believe that a disease so widespread as this, which begins at the same period of

life, and which involves several members of the same family and leaves others exempt, could be due to any toxic influence that is known to us. As an argument against the theory that the disease was carried through the mother's milk, the speaker cited his first two cases, who, although Jewish children, were not raised on mother's milk.

Dr. Sachs said that, personally, he had no theory to offer regarding the causation of this disease. He thought it was in line with many of the hereditary diseases, and we must accept it as a hereditary family disease of the central nervous system. It certainly bears a close clinical relationship to other hereditary family affections, particularly to those characterized by blindness, for example, or to those characterized by spastic paraplegia and forms of mental defect.

The evidence furnished by Dr. Hirsch of involvement of the gray matter of the spinal cord explains why in some cases the paralysis is spastic and in others flaccid (as in Sachs' first patient). In conclusion, Dr. Sachs said that, while he regarded this as a widespread disease of the central nervous system, he did not believe that the spinal cord need always be affected, and that in some instances the disease may be chiefly cerebral.

Dr. Carl Koller had seen five cases of this interesting affection. Two cases, which he had published a few years ago in a paper read before the American Ophthalmological Society, had been the immediate occasion of this question being taken up again by Dr. Sachs. A number of cases had been reported in quick succession, and a thorough study made of it clinically by Dr. Sachs, and later, pathologically, by Dr. Hirsch, whose painstaking and exhaustive research had greatly contributed to our understanding of the disease.

Dr. Koller said that the characteristic ophthalmoscopic changes in the macula were not congenital, and were not found until between the fifth and seventh months. In two of Kingdon's cases, which were examined in the earlier months of life, they were missing, and in two cases of the speaker, which were examined in the first two months of life, they were also absent. One of the latter was the identical case which Dr. Peterson had just reported in his paper. The speaker said he had examined that child five times between the eighth and sixteenth weeks of its life, the ophthalmoscopic examination being negative, although other unmistakable signs were present which proved that the case belonged to this class. In the other case, the characteristic changes developed later.

Dr. Koller thought that the atrophy of the optic nerve in these cases made its appearance considerably later than the changes in the macula, and developed gradually. As to

whether the former depends on the latter, or whether one was independent of the other, that point remained for pathologists to determine, but it seemed reasonably certain that, as a rule, the ocular changes were not congenital.

Another question arising in connection with this subject was whether these patients ever had sight, in our meaning of the word; whether they were born blind, or with a vision already impaired by existing cerebral changes; or whether they were born with a vision equal to the low degree pertaining to the newborn, with degeneration setting in before the vision became developed to a higher plane. At all events, the behavior of the children with regard to their vision was not normal at the time when no ophthalmoscopic changes were visible, and the conclusion was permissible that vision was impaired by cerebral degeneration lying pretty far back. In one of the speaker's cases the mother had her suspicions in regard to the child's eyesight as early as the fourth week of life. In the two cases he had examined in the 8th and 9th weeks he had received the impression that vision was already impaired. The light of a candle in a dark room was not followed by the eyes, and no prompt reaction of the pupils could be produced. The pupils became slowly narrower when exposed to light, and slowly dilated in the dark. The speaker thought these two facts certainly deserved weight, considering the difficulty which exists as to how much vision such young children enjoy.

Dr. Charles K. Mills concurred with Dr. Sachs in regard to the nature of amaurotic family idiocy. The word "congenital" in connection with these cases seemed to him to be sometimes misused. In many of these cases the clinical, and, possibly, the pathological, evidences of the disease, so far as can be demonstrated, are not observed until weeks, months, years, or even decades, after the birth of the individual, and in this sense they are not congenital; but these cases, along with some of the forms of diplegia and of muscular atrophy, and perhaps of Huntington's chorea and developmental insanities, belong to a great embryonal class. The potentiality of development in these cases is limited, and it may be weeks, months or years before the symptoms make their appearance. Whether the immediate pathological conditions which initiate the manifestations of the disease are toxic or non-toxic, sooner or later the degenerative process will manifest itself, if the individual survives long enough.

Dr. F. X. Dercum said that he had reported one family in which three of the four children suffered from spasmodic diplegia and idiocy. Sachs, Freud, and others had reported similar instances. In his three cases the symptoms of the

degenerative process manifested themselves in each instance immediately after measles; the only child that escaped did not contract measles. Such observations as these, Dr. Dercum thought, tended to strengthen the toxic theory of amaurotic family idiocy.

Dr. W. G. Spiller reported the case of a child, born of healthy parents, but who had an aunt insane and an uncle who was feeble-minded. The child had presented no symptoms detectable by the parents until he went to school, when he was noticed to be somewhat feeble mentally. His gait and speech were rather slow; he lisped slightly, and his circulation was imperfect. As time went on, spastic symptoms developed, and gradually became very marked; the reflexes were much exaggerated, intention tremor was noticed, the speech became scanning, and was finally lost. The later developments were incoördination, feeble mentality, muscular contractures, intense atrophy, and finally complete paralysis. When the boy was thirteen years old, bilateral optic atrophy was noted, but it may have existed for some years before the examination was made. The child died at the age of fifteen. A microscopical examination showed distinct degeneration of the pyramidal tracts of the cord. The cells of the anterior horns and the spinal roots appeared to be little altered. The brain could not be obtained.

Dr. Spiller said he did not consider this case a typical one of amaurotic family idiocy, and yet it bore certain resemblances to this disease. It seemed to him to have a closer relation to the pseudo-sclerosis of Westphal and Strümpell, for the pyramidal tracts were not normal in all instances. As only five cases of this pseudo-sclerosis are now on record, Dr. Spiller was inclined to regard his case as a sixth. The report will be published in detail later.

Dr. Hirsch, in closing, said he did not think we could dispense with the distinction between a disturbance of development and an acquired disease. To accept the statement that an arrest of development is identical with an acquired disease would mean to destroy wilfully the most valuable scientific work of the last decades. In the nervous system, as elsewhere in the body, a certain predisposition to disease may be inherited; tuberculosis, for example, although a strong predisposition to it may be inherited, cannot develop without the presence of the tubercle bacilli. The speaker said he did not question the fact that children who develop amaurotic family idiocy were born with a normal nervous system. If the changes in the macula lutea are produced by changes in the ganglion cells, we must assume that as long as the former changes are absent the ganglion cells are normal, and it is

to-day a clinically established fact that the children are not born with the peculiar condition in the macula lutea, but that this develops during the course of the disease. In his case the optic tract was atrophied; this atrophy was secondary; it was an acquired condition; the child was not born with it, but had impaired vision up to the age of eight months.

Dr. Hirsch said he agreed with the previous speakers that the toxic origin of this disease was still an open question. As regards the milk theory, he did not think it was disproven by the instance cited by Dr. Sachs, where two Jewish children, who afterward developed the disease, had been brought up by Christian wet-nurses. From the fact that all the cases which have been observed so far belonged to the Jewish race, it does not follow that the toxin must also necessarily be of Jewish origin.

The speaker said he wished to take exception to the statement that amaurotic family idiocy was in line with other hereditary diseases. He knew of no other inherited family disease where all the nerve cells, and nothing but the nerve cells, in the body became affected; the condition, so far as he knew, had no analogy in pathology. Furthermore, in this disease the degenerated cells had a typical appearance, only analogous to what we see in poisoning, and that, in spite of this, they had certain characteristics of their own, was in accordance with the modern view that every poison had a specific action on nerve cells.

-
167. DELIRE DE PERSECUTION; KYSTE DERMOÏDE DE L'OVAIRE; LAPAROTOMIE; DISPARITION DES TROUBLES MENTAUX (Delirium of Persecution; Dermoid Cyst of the Ovary; Laparotomy; Cessation of the Mental Trouble). A. Voisin (*La France Méd. et Paris Méd.*, Feb. 12th, '97).

Under this title the author publishes the following case: The patient, with a neuropathic family history, had a pessary introduced for prolapsus uteri, six years ago. Being a virgin, this treatment made such an impression on her as to disturb her mental faculties, and she became sad and at times showed violent temper. Later on she developed marked delirium of persecution, with hallucinations of sight and hearing, accused several persons of plotting against her, and tried to commit suicide twice. In November last, laparotomy was performed, and a large multilocular ovarian cyst extracted. For two days after the operation the patient was somewhat excited, then all the cerebral disturbances ceased permanently. The foregoing observations of mental disturbance, being cured by a surgical operation, is considered as extremely rare and interesting, inasmuch as one is accustomed to observe mental symptoms develop subsequent to surgical interference upon the female genital organs.

MACALESTER.

Periscope.

With the Assistance of the Following Collaborators:

CHAS. LEWIS ALLEN, M.D., Wash., D.C. R. K. MACALESTER, M.D., N.Y.
J. S. CHRISTISON, M.D., Chicago, Ill. J. K. MITCHELL, M.D., Phila., Pa.
A. FREEMAN, M.D., New York. H. PATRICK, M.D., Chicago, Ill.
S. E. JELLIFFE, M.D., New York. JOSEPH SAILER, M.D., Phila., Pa.
WM. C. KRAUSS, M.D., Buffalo, N.Y. HENRY L. SHIVELY, M.D., N.Y.
W. M. LESZYNSKY, M.D., New York. A. STERNE, M.D., Indianapolis.

ANATOMY AND PHYSIOLOGY.

168. NOTE ON "THORNS" AND A THEORY OF THE CONSTITUTION OF GREY MATTER. A. Hill (Brain, 20, 1897, p. 131).

"Granules," or if one prefers Cajal's term, "thorns," have been supposed to be dots of naked protoplasm, by means of which the dendrites establish connections with the terminal end brushes of nerve fibres. The author here presents a brief note, in which he states that they are universally present but exist in a variety of forms, four of which he details. He believes them to be structures imperfectly revealed by any of our known methods. The great variation in form leads him to the belief that a "thorn" is really the cell end of an unstainable nerve filament surrounded by a film of staining cell plasm." The author's brief notes with reference to the nervous mechanism are of interest in view of Apáthy's recent observations respecting the continuity of fibrils and their arrangement. JELLIFFE.

169. ACTION DE LA LUMIÈRE COLORÉE SUR LA RETINE. (Action of Colored Lights on the Retina). E. Pergens (Annales de la Soc. Royale des Science Médicales et Nat. de Bruxelles, 1897, 6, p. 1).

The results of the author's studies on the action of monochromatic lights on the movements of pigment and the morphologic-histo-chemical changes in the nervous cells of the retina were substantially as follows:—

The migration of pigment is at its maximum for the blue, at a minimum for red. Thus it is not the intensity of the light which causes this migration, as physics show, that red light is more intense than blue.

When a single eye is illuminated the pigment in the retina of the closed eye also migrates, varying with the character of the light. The contraction of the cones following the action of the various spectral portions of light varies. The quantity of the nuclei contained in the rods and cones diminishes under the action of the rays of the spectrum but not in a degree corresponding with the luminosity. This decrease is at its maximum for the red, and at a minimum for the green.

Basophil structures in the cells were more acted upon than acidophil or neutrophil portions. The action of the X rays was negative.

ELY.

170. DE L'IMPORTANCE FUNCTIONELLE DU CORPS CELLULAIRE DU NEURONE (On the Functional Importance of the Cell Body of the Neuron). C. A. Pognat (*Revue Neurologique*, 6, 1898, p. 158).

The author presents a brief summary of the ideas of Cajal, van Gehuchten, and Lugaro, with reference to the relation of the cell body to the dendritic and axis cylinder processes of the nerve cells. He discusses Cajal's so-called laws, and maintains that they are open to much well-founded criticism. The author himself holds that the neuron is a nervous cellular unit, possessing two types of conductors, protoplasmic processes and the axis cylinder process, but it also possesses a centre, which is genetic, trophic and functional, which is nothing else but the cell body.

JELLIFFE.

171. SOME JUDGMENTS OF THE SIZE OF FAMILIAR OBJECTS. H. K. Wolfe (*American Journal of Psychology*, 1898, 9, p. 138).

The author experimented on nearly eleven hundred persons with coins and currency bills. The following are his more important deductions:—

1. Young children underestimate the size of coins and bills.
2. Mature persons of intelligence overestimate the size of the silver dollar, half dollar and quarter dollar.
3. All classes of persons underestimate the size of the dime, nickel and bill.
4. Girls overestimate their coins and their equivalent squares more than boys do. In other problems of this investigation the boys made the larger figures.
5. The judgments of the eighth-grade children were more uniform than the fourth-grade or university students.
6. Within the same class age causes no appreciable effect.

CHRISTISON.

172. A GRAPHIC STUDY OF TREMOR. A. A. Eshner (*Jour. of Experimental Med.*, 2, 1897, p. 301).

The author, in an extended series of observations, studies the following questions: 1. Whether or not a demonstrable tremor exists in healthy individuals; 2. whether or not any relation or gradation exists among various kinds of tremor; and, 3. whether or not various forms of disease show, as to their tremor, distinguishing characteristics. The summary of the conclusions reached is as follows:

1. All muscular movements are made up of a series of elementary contractions and relaxations, which may be appreciable as tremors in conditions of both health and disease.
2. The differences between different tremors are of degree rather than of kind, i. e., no form of tremor is distinctive of any one disease or group of diseases.
3. No definite relation exists between one form of tremor and any other.
4. The frequency of movement is in inverse ratio to the amplitude, and vice versa.
5. Habitual movements are performed with greater freedom from tremor than unusual movements.
6. There is no material difference between the movements of the two sides of the body, except as related to proposition 5.

The paper is well illustrated with tracings and the apparatus used well described.

JELLIFFE.

PATHOLOGY.

173. SUR LES MODIFICATIONS HISTOLOGIQUES DES CELLULES NERVEUSES DANS L'ÉTAT DE FATIGUE. (The Histologica Modifications in Nerve Cells during Fatigue). A. M. Puguat (La France Méd., 44th year, p. 745).

The studies of different authorities on the modifications that nerve cells undergo, in different stages of fatigue, have yielded contradictory results up to the present time, and the author enumerates his observations of histological changes in the spinal ganglia of young cats, which are best adapted to experimentations and the preparations of nerve fibres, to which the electrodes are applied. These latter are placed at a distance of three or four cms. from the ganglion, in order to avoid any mechanical irritations by the electrical current, and it is generated by a Leclanché cell with induction coil. The manifestations of fatigue observed in nerve cells are a diminution of the cellular volume and its nucleus, and disappearance of the protoplasmatic chromatine substance. As the stimulation is kept up the chromatine granules become less visible by degrees, disappearing to a great extent at the end of about sixteen minutes, when they are seen only in the periphery of the cell in an annular arrangement. When the stage of exhaustion is reached—about twenty-four minutes of stimulation—the chromatine granules are entirely absent in the cytoplasm, which latter takes on a pale uniform tone, the nerve cell contracting in a manner as not to fill its reticulum completely, and the nucleus diminishing in size, with indistinct outlines. Other changes found in the nucleus, or its emigration towards the periphery of the cell, described by others, are not confirmed by the author. In regard to the two principal factors concerned in the experimental fatigue of nerve cells, viz: the intensity and duration of stimulation, the former is of paramount importance, it proving that a strong current, acting but a short time, causes much more marked cellular changes than a weaker one of longer duration.

MACALESTER.

174. RÜCKENMARKSBEFUNDE BEI GEHIRNTUMOREN (Findings in the Spinal Cord in Cases of Brain Tumor). Josef Ursin (Deutsche Zeitschrift für Nervenheilkunde, 11, 1897, p. 169).

Ursin reports three cases of brain tumor with changes in the posterior columns of the spinal cord. In two of these the degeneration was of an ascending type, and due to involvement of the posterior roots, but in the third it was evidently primary, for the posterior roots were degenerated at their entrance into the cord only in the upper part of the cervical region. Changes in the ganglion cells of the cord were noted in all three cases, but only in one was the extraspinal portion of the posterior roots altered. Meningeal involvement was not observed in any of the cases.

Two views are held regarding the alteration of the posterior columns in cases of brain tumor: (1) the degeneration results from increased pressure of the cerebrospinal fluid (Mayer), and (2) that the degeneration occurring in cases of malignant growths is analogous to what is seen in the peripheral nerves in such conditions (Dinkler).

If Mayer's view were correct, the degeneration of the posterior roots would be greatest where the pressure is greatest, i. e., in the lower parts of the spinal cord, but in a number of cases the degeneration has been found to be most intense in the cervical region, and in others no signs of increased intracranial pressure have been noted, or where these signs have been present the degeneration of the posterior roots has been comparatively slight. Mayer's view also does not

explain the greater involvement of the roots of one side of the cord. In reply to Mayer's statement that if the toxic theory were correct, the extraspinal portion of the posterior roots would be the first to suffer, inasmuch as this part is surrounded by the cerebrospinal fluid, but that actually in cases of brain tumor this portion of the roots is relatively intact, Ursin refers to the well-known cases of toxic degeneration of the posterior roots as seen in pellagra, ergotism, diabetes, etc., in which the extraspinal portion of the posterior roots was not degenerated, and which resemble in this respect the cases of brain tumor.

Ursin believes that the lesions in the spinal cord in cases of brain tumor may be an intramedullary, primary degeneration of the posterior columns, changes in the ganglion cells, and changes in the remaining white matter, and in addition to these lesions the extramedullary portion of the posterior roots may be affected. The cause is to be found in intoxication and malnutrition. SPILLER.

175. DETAILED REPORT UPON THE CLINICAL AND PATHOLOGICAL FEATURES OF SIX CASES WHICH CAME TO AUTOPSY. A. N. Ohlmacher (Bulletin of the Ohio Hospital for Epileptics, January, 1898, p. 4).

Six cases of epilepsy are here reported upon with more than usual attention to details; they form the basis of a second paper of the author, "Upon the Resemblance of the Foregoing Cases of Epilepsy to Certain Diseases Associated with Thymic Hyperplasia," p. 43, in which the author discusses:

1. Thymic asthma.
2. Sudden death in adults with persistent thymus.
3. Exophthalmic Goitre.

The author shows that in four of the cases of epilepsy there was a marked increase of the thymus, and he further adds that while four cases do not form a basis for weighty conclusions, still, when these four cases are of a disease in which the morbid anatomy has always been dark; in which a constant gross lesion in even four consecutive cases has been almost unknown; and in which, unfortunately, attention has been almost exclusively centred upon the brain,—then the discovery of a uniformly characteristic condition, outside of the brain, even in four cases, carries with it a hopeful suggestion. Further, when it happens that the peculiar morbid anatomy fits in with several other conditions in which certain clinical analogies can be shown, and particularly when these conditions are almost as mysterious as epilepsy, then it seems justifiable to direct careful attention to the various relations suggested by the study.

"One thing must be certain, and that is, that somewhere, somehow the peculiar morphological anomalies found in our few cases of epilepsy and also noted in thymic asthma, thymic sudden death and possibly Basedow's disease, will be found to have more than mere accidental bearing, for assuredly it is not nature's habit to leave behind in a certain unfortunate class of human beings a series of morbid anatomical conditions, such as those we have considered, without some weighty purpose behind her."

JELLIFFE.

176. BACTERIOLOGIE DE DEUX CAS DE CHORÉE AVEC ENDOCARDITE (Bacteriology of two Cases of Chorea with Endocarditis). M. Apert (La Med. Moderne, 8, 1898, p. 80).

M. Apert, Soc. de Biologie, in two cases of chorea with endocarditis observed in the service of M. Dieulafoy, made cultures from the blood in milk, by the procedure suggested by Thierloix. In the first case, in which the chorea was already disappearing, the cultures re-

mained sterile. In the second, a chorea at its height, a diplococcus of granular, oval shape without a capsule was observed, staining with Gram, apparently identical with that described by M. Triboulet in cases of acute articular rheumatism.

It should be added that both the chorea patients upon whom the investigations were made, had had alternating attacks of chorea and acute rheumatism.

MITCHELL.

177. ON CERTAIN CHANGES IN THE CELLS OF THE VENTRAL HORNS AND OF THE NUCLEUS DORSALIS (CLARKII) IN EPIDEMIC CEREBRO-SPINAL MENINGITIS. Lewellys F. Barker (British Medical Journal, ii., 1897, p. 1839).

The author describes two kinds of alteration. First, slight changes in the cells of the anterior horns, such as occur from various poisons, and which he attributes to the toxæmia of the disease, viz.: (1) the disappearance of the stainable substance of Nissl from the dendrites or from portions of the dendrite or of a cell body; (2) the formation of nodular swellings of the dendrites, these swellings corresponding to pathological accumulations of the stainable substance; and (3) a tendency to disorganization of individual Nissl bodies, especially at the periphery of the cell.

Second, lesions not at all similar to the first, but practically identical with those which take place in the cell body of a neuron after an injury of the axon which belongs to it. These latter changes were found in the cells of the anterior horns and in those of Clarke's columns. The alterations in the anterior-horn cells are attributed to the involvement of the anterior nerve roots in the meningeal inflammation; those in the cells of Clarke's columns, not to an affection of the posterior roots, but to the damage done to the direct cerebellar tracts. The meningitis was particularly intense at this part of the periphery of the cord, and the fibres of this tract are supposed to be neuraxons of the cells of Clarke's columns.

PATRICK.

178. DIE COLLOIDENTARTUNG DES GEHIRNS (Colloid Degeneration of the Brain). A. Alzheimer (Archiv f. Psychiatrie, 30, 1898, p. 19).

The author describes two cases of colloid degeneration. The first occurred in a case of general paresis with optic atrophy and characteristic convulsive attacks. The sections showed leptomeningitis and pachymeningitis and chronic colloid degeneration of the large ganglion cells of the cortex.

A second case showed unilateral convulsions, loss of memory and stupor, and finally hemiplegia and coma.

The autopsy showed colloid degeneration of the convolutions of the right hemisphere and basal ganglia, with secondary softening in the basal ganglia and crura.

A chemical and micro-chemical study of the colloid substance in both cases showed the following characters. It is soluble when fresh in warm water; with picro-carmin, or with double staining with carmine and hæmatoxylin the colloid substance colors a decided red, especially in specimens hardened with bichromate. With Van Gieson's mixture colloid stains a light red, distinguishing it from hyaline substance. Eosin stains it deep red, and Rosin's mixture, which is to be specially recommended, stains colloid flesh red, nuclei bluish green, blood cells yellowish red and the rest of the tissue a light red. Weigert's fibrin staining methods also give good differential stains.

The best results were obtained with bichromate hardening. Alcohol is not good for colloid. The vessels were not affected in the degeneration. The paper is well illustrated and is a noteworthy con-

tribution to our knowledge of a type of tissue degeneration and an excellent discussion of the micro-chemical means we possess whereby one may differentiate the many closely allied forms. JELLIFFE.

CLINICAL NEUROLOGY:

179. TUMOR OF THE MENINGES IN THE REGION OF THE PITUITARY BODY PRESSING ON THE CHIASMA. J. W. Sterling (*Annals of Ophthalmology*, 6, 1897, p. 15).

The author reports a case of this character. The patient was a man forty-two years of age with total blindness of the left eye, and complete right hemianopsia of the right eye. There was a history of alcoholism, but none of syphilis. There was optic atrophy. Both knee-jerks were slightly exaggerated. No motor or sensory disturbances. Frequent flushings and perspiration of the head and back of the neck occurred. The further course of the case was very slow, six years elapsing before death. In about eighteen months he was totally blind. He became fat and flabby, always had a good appetite, and was greatly troubled with continuous sleepiness. The post-mortem examination revealed a tumor as large as a hen's egg growing from the meninges and pressing on the front of the chiasma. Microscopically it proved to be an endothelioma.

LESZYNSKY.

180. A CASE OF BULLET IN THE LEFT HEMISPHERE OF THE BRAIN SHOWN BY SKIAGRAPH. James Bell (*Annals of Gynecology and Pediatrics*, 10, 1897, p. 353).

A little girl, four and a half years old, accidentally discharged an English "Bulldog" revolver, one bullet entering her brain. The bullet entered the forehead over the centre of the left orbit, and rather less than half way from the margin of the orbit to the edge of the hairy scalp. She did not lose consciousness, nor show any signs of special suffering. She was sent to the General Hospital, where she was chloroformed, and an attempt to remove the foreign body was made. Four fragments of bone were removed from the brain substance—one of them from a depth of an inch and a quarter from the dura mater. Some dark blood clots and brain detritus escaped alongside of the forceps. The operation was attended with failure, as at each attempt to grasp the bullet with the open blades of a forceps it receded further into the cerebral substance. All efforts were finally abandoned. The wound was allowed to heal by drainage and granulation. The patient suffered no ill effects from the operation, and made a rapid recovery. The child since being discharged from the hospital never suffered from headache or any other brain symptom. She was always a bright child, but her parents consider her even brighter than before the accident.

In August, 1896 (the accident occurred in January, 1894), the little girl was skiagraphed by Professor Callender, of McGill University. The skiagraph distinctly showed the bullet in the brain. Plates accompany this report. ABRAHAM.

181. THE OCULAR MANIFESTATIONS OF INTRACRANIAL TUMOR. Martin (*Lancet*, July 10th, 1897, p. 81).

In an editorial discussion of the work of the author, based on an analysis of six hundred cases of intracranial tumor the *Journal of the American Medical Association* (September 4th, 1897) summarizes the results as follows. The distribution of the new growths was: Cerebellum, 138; motor area, 120; frontal area, 61; pons and medulla, 45;

parieto-occipital area, 37; basal ganglia, 36; centrum ovale, 30; temporo-sphenoidal area, 28; pituitary, 26; general, 19; corpora quadrigemina 15; corpus callosum, 13; crura, 5. The nature of the growths was as follows: sarcoma, 126; glioma, 82; tuberculous, 72; cystic, 44; gumma, 36; gliosarcoma, 30; hydatids, 21; carcinoma, 11; other varieties, 179. Of all cases of intracranial tumor 68.8 per cent. occur among males. Headache is more likely to be absent in connection with tumors of the motor area and of the corpus callosum than in connection with tumors situated elsewhere. The tumor is generally situated on the side on which internal strabismus is noted. Only limited localizing value is conceded at present to optic neuritis, but the hope is expressed that improved methods of examination now in course of adoption will give it greater value in the future. When a difference in degree of optic neuritis in each eye exists, it is more than twice as probable that the tumor is on the side on which the neuritis is the more marked. It should further be borne in mind, 1. That optic neuritis is constantly present in association with tumors of the corpora quadrigemina. 2. That it is present in 89 per cent. of cases of cerebellar tumor and of tumor of the posterior part of the cerebrum. 3. That it is absent in nearly two-thirds of the cases of tumor of the pons and medulla and of the corpus callosum. 4. That it is least frequently met with in cases of tuberculous tumor, and is most common in cases of glioma and cystic tumor. SHIVELY.

182. DIE AMAUROTISCHE FAMILIÄRE IDIOTIE (The Amaurotic Family Idiocy). B. Sachs (Deutsche medicinische Wochenschrift, 24, 1898, p. 33).

Sachs states that twenty-seven cases of this interesting form of idiocy have been reported. The children afflicted with this disease appear to be normal during the first weeks of life, but after two to eight months they take less interest in their surroundings, become unable to sit up or hold up their heads, and make few voluntary movements. Failure of sight is noticed after some months. Weakness of the extremities becomes spastic paralysis, but may be flaccid. Convulsions are rare. The reflexes may be exaggerated, normal or diminished. Blindness, due to changes in the macula lutea and optic atrophy, is usually total at the end of the first year, by which time the child is also idiotic. The disease attacks several children in the same family. Occasional symptoms are nystagmus, strabismus, hyperacusis or deafness. Macular changes have been found in all the reported cases.

In the two cases examined by Sachs degenerative changes were found in the large pyramidal cells and fibres of the cerebral cortex. The vessels were normal, and there were no signs of inflammation. Degeneration of the crossed motor tracts was noticed in one case, though this may possibly have been arrested development of these tracts. The ætiology is unknown, but the disease is not due to syphilis. SPILLER.

183. CENTRALE BEIDERSEITIGE AMAUROSE INFOLGE VON METASTATISCHEN ABSCESSEN IN BEIDEN OCCIPITALLAPPEN OHNE SONSTIGE HERDSSYMPTOME (Bilateral Central Amaurosis Resulting from Metastatic Abscesses in Both Occipital Lobes Without Other Focal Symptoms). H. Heinersdorff (Deutsche med. Wochenschrift, 23 1897, p. 230).

Cases of bilateral amaurosis from lesions in both occipital lobes are uncommon, and, according to Heinersdorff, all the cases which have been reported have been due to softening of the cerebral sub-

stance in consequence of emboli. The writer reports a case which he states is the only one of its kind in the literature. A man became completely blind within fourteen days, and suffered from transitory headache and fever. An abscess developed in the groin. The pupils reacted distinctly though slowly to light. The eyegrounds, the urine, the power of motion and sensation were normal. The patient was somewhat somnolent, and died in coma a few weeks after the loss of sight. At the autopsy an abscess was found in the region of the liver, and one in each occipital lobe. The lateral ventricles were filled with pus. The cerebral abscesses were supposed to have been metastatic.

SPILLER.

184. A CASE OF TUMOR OF THE OBLONGATA. H. S. Upson (*Annals of Ophthalmology*, 6, 1897, p. 136).

The author reports the case of a girl seven years of age, who was quite well until she was three years old. Then she began to have convulsions, consisting of loss of consciousness, frothing at the mouth and general rigidity, but there were no clonic spasms. Within a month after the onset she began to lose power in the left hand, and later in the left leg. No convulsions in last three years. Very little headache was complained of. Right facial paralysis with lagophthalmos, right convergent strabismus with paralysis of the external rectus. Left upper extremity paralyzed. Walking impossible—the feet cannot be moved at all, but the leg can be bent at the knee rather feebly. No sensory disturbance. All reflexes exaggerated on the left side. No ankle-clonus. No tenderness on percussion over the skull. Pupils normal. No optic neuritis. Later she was unable to sit up. Difficulty in mastication and deglutition, and double optic neuritis.

LESZYNSKY.

185. TUMOR (GLIOMA) OF THE LEFT TEMPORAL LOBE OF THE BRAIN; ATTEMPTED REMOVAL. M. Allen Starr and R. S. Weir (*Medical News*, 11, 1897, p. 170).

The authors report an interesting case of brain surgery. The patient, a woman *æt.* 55, showed a slowly progressing motor aphasia, to which was added, after three months, a rather rapidly increasing right hemiplegia. The localization was therefore made of a tumor in the left third frontal convolution growing backward and inward, so as to compress the motor tract in its passage toward the internal capsule. On this diagnosis an operation was undertaken, but it failed to reveal any tumor in the part exposed. The patient, of course, was not relieved of any of her symptoms. The wound healed perfectly without suppuration, the sutures being removed on the fourth day, and her pulse was quite regular and natural, but she gradually sank into a comatose condition, the hemiplegia becoming absolute and the aphasia total. She died quietly on the eighth day after operation. At the autopsy the tumor, an infiltrating vascular glioma, was found deep within the apex of the temporal lobe. It must from its location have produced pressure inward and upward, as any pressure downward or outward was prevented by the walls of the skull. Such pressure naturally affected the function of the parts compressed, and as these were the third frontal convolution and the island of Reil with the motor tracts beneath it in the capsule, the symptoms were necessarily misleading.

SHIVELY.

186. EIN FALL VON TUMOR DER INNEREN KAPSEL. (Brain Tumor). Jacobson (*Centralbl. f. Nervenheilkunde*, 8, 1897, p. 244).

The author reports a case of cerebral tumor somewhat unusual in several aspects.

The patient, a child of 5, while confined to the bed with a series of acute diseases, gradually developed spastic weakness of the left side with some anaesthesia. The paresis continued to increase after the patient was out of bed and constituted the only symptom, except moderately choked discs, and toward the last a few attacks of faintness without loss of consciousness. The choked discs completely disappeared and did not return, and because of this and the inconspicuous general symptoms, the author was inclined to diagnose a focus of cerebral softening rather than tumor. The autopsy revealed a tumor, about the size of the thumb, in the left cerebellar hemisphere, and another, as large as a small apple, in the right cerebrum that destroyed the entire lenticular nucleus, most of the posterior limb of the internal capsule, and part of the optic thalamus. Both were solitary tubercles.

The author explains the absence of severe general disturbance, as well as spontaneous disappearance of the choked discs, by the fact that the bones of the cranial vault had become very thin and elastic, allowing them to bulge, thus in some degree preventing great increase of intracranial pressure. He does not consider, however, that the disappearance of choked disc from relief of pressure is absolutely conclusive proof of the mechanical pressure as opposed to the toxic cause of this condition, as an operation diminishing intracranial pressure may be conceived to allow of the re-establishment of a natural circulation in the lymph channels, which permits the removal of toxic agents that presumably cause the optic neuritis.

In the discussion Oppenheim confirmed the disappearance of the choked discs in the present case in spite of the growth of the tumor, and was inclined to favor the mechanical origin of the optic neuritis.

Schuster also reported the disappearance of choked disc in a tumor case. A young woman who presented all the principal symptoms of tumor was put on inunctions of mercury and large doses of potassium iodide, whereupon the headaches ceased and the choked disc disappeared. Some months later she suddenly died, and the autopsy revealed in the left posterior fossa a glioma, the size of a hen's egg, which showed no trace of any action of the iodide.*

Greeff thought that clinically as well as pathologically a difference should be made between pure choked disc (passive congestion) and optic neuritis. The latter means severe change in the nerve fibres; the former may exist to a marked degree without damage to the optic nerve and with normal vision and visual fields. PATRICK.

187. GLIOM DER MEDULLA OBLONGATA. (Glioma of the Medulla Oblongata). J. Collins (*Deutsche Zeitschr. f. Nervenheilk.*, 10, 1897, p. 453).

The first symptom noticed in this case was paræsthesia in the fingers of the left hand and later in the left upper and left lower limbs. Incoördination of the left hand and left leg, paræsthesia in the left side of neck and occipital region, insecurity in the standing position with inclination toward the right side, exaggeration of both patellar reflexes, ankle clonus of the right side with some diminution of motor power on this side of the body, almost complete analgesia of the body, and especially on the left side, with preservation of tactile sense were noted. The cardinal symptoms of brain tumor—optic neuritis, vomiting, vertigo, headache, etc., were absent. There were, therefore, no distinct localizing symptoms. At the autopsy the oblongata was found to be asymmetrically increased in size, and a new growth pro-

* A similar case has been seen by the reviewer who referred the good effects of the iodide to the relief of the secondary œdema, and hence the pressure, without any change in the growth itself.

jected from the dorsal aspect into the fourth ventricle. The oblongata was a mere shell about the tumor. Microscopic examination showed that the growth was a glioma, and that it had in large part destroyed the oblongata, although the symptoms had been rather indefinite and had lasted nearly two years. SPILLER.

188. A CASE OF CEREBRAL URÆMIA WITH CATALEPTOID ATTITUDES. E. J. Kempf (American Practitioner and News, 23, 1897, p. 40).

A woman, aged 29, had convulsions in her first three confinements. The fourth, fifth and sixth labors were normal. In the seventh, convulsions again came on just as the first stage of labor was about completed, and lasted for several hours. Afterward the patient became quiet for several days; everything was normal. On the fourth day she again had convulsions. During the next two weeks a difficulty of breathing developed gradually into the peculiar rhythm of Cheyne-Stokes respiration. There was no visceral or pelvic inflammation, and no odor to the lochia. The temperature oscillated between 97 and 104° F. At times there were vomiting, double vision and hallucinations. The amount of urine varied between 16 and 35 ounces, the quantity of albumin between 1 and 16 grains per ounce and the total urea excreted amounted to from 200 to 400 grains in the twenty-four hours. The urine also contained mucous, epithelial and hyaline casts. At times she would walk without much effort and seemed to possess full consciousness; then without any premonition she would pass into a state of hebetude; on lifting her hand it was noticed that the joints were rigid and the arm remained in the position in which it had been held, but gradually descended by its weight to its place on the bed. The legs were in the same condition as the arms. In spite of the rigidity and delusions, she had not lost all consciousness but was in a state of hebetude from which she could be aroused for a few seconds. When she recovered full consciousness, she described some of her impressions and talked about her delusions. It was not therefore a case of true catalepsy. The convulsions were of a tonic type and of a cataleptoid character, and the cerebral symptoms were evidently of the nature of uraemic manifestations. The patient improved slowly and though still an invalid is now able to be up and do light housework. The cataleptoid condition described in this case is a phenomenon only discovered when carefully looked for. In this patient it lasted for nearly three weeks. The cerebral phenomena are to be attributed to certain pathological changes due to the retention of effete products. An interesting point in the case is that a patient can have four distinct attacks of puerperal convulsions and continue to live. Another interesting feature was the recurrence first of puerperal convulsions of a clonic form with apparent recovery and after a few days the cataleptoid condition, lasting three weeks and followed by apparent recovery. It is plausible to say this is one of those cases formerly classed under the title of hysteria, but which more and more are being understood as due to a poisoned condition of the system caused by defect of the assimilative or excretory organs. FREEMAN.

189. ZWEI FÄLLE VON TABES DORSALIS MIT SPERMINUM-POEHL BEHANDELT (Two Cases of Tabes Treated with Sperminum-Poehl). M. Werbitzky (Deutsche Med. Wochens., 23, 1897, p. 67; Therapeutische Beilage).

The author concludes that the treatment has resulted in increase of all forms of sensory impressions, with diminution of pain and improvement of the general tone. There was increase in muscular strength and muscular sensibility, diminution of the ataxia, and an improvement in the eye symptoms. VOGEL.

THE
Journal
OF
Nervous and Mental Disease

Original Articles.

CONGENITAL FACIAL PARALYSIS.*

By H. M. THOMAS, M. D.,

Clinical Professor of Neurology in the Johns Hopkins University; Neurologist of
the Johns Hopkins Hospital.

For the last few years a great deal of interest has been taken in congenital abnormalities of muscles. The absence of one or more muscles, or parts of muscles, has been noted from time to time, and although such a congenital absence is by no means common, still a considerable number of cases has been reported.

The pectoral muscles seem to be particularly prone to such a defect in development, as by far the greatest number of reported cases has to do with these muscles. Hoffman,¹ from Eichorst's clinic, and Kalisher² have collected and analyzed these cases. Many of the cases showed other congenital abnormalities, such as malformations of the skin and hair, webbed fingers, etc., etc.

Möbius,³ in 1888, and again in 1892, called attention

*Read before the American Neurological Association, May, 1898.

¹ Hoffman: Virchow's Archiv., vol. cxlvi., 1896, p. 163.

² Kalisher: Neurolog. Centralbl., vol. xv., 1896, pp. 685, 732.

³ Möbius: München. med. Wochenschr., xxxv., No. 6, 7, 1888; ibid., xxxix., No. 2, 4, 1892

to an interesting group of cases, in which there was a congenital defect in the movements of the eyes, combined, at times, with a similar defect in the facial muscles. With these he associated certain cases which developed in infancy or childhood, and called them all infantile nuclear atrophy (infantilen Kernschwund), for he believed that the process was essentially the same in all the cases—a degeneration and atrophy of the nuclei of the cerebral nerves.

The muscles of the eye were affected in all of the cases that Möbius collected. That the facial muscles might be affected alone, he thought was probable, although he had been unable to find any record of such cases.

Schultze,⁴ in 1892, and Bernhardt,⁵ in 1894, report cases of unilateral congenital facial paralysis. I shall refer to these cases again.

Kunn,⁶ of Vienna, in 1895, in a monograph considers the congenital defects in the movements of the eyes. (*Die angeborenen Beweglichkeitsdefecte der Augen.*) He has abstracted and tabulated all the cases he could find in literature. Among these 73 cases, there were 11 in which the facial muscles were also involved. He refers to several cases of uncomplicated congenital facial muscular defect. As opposed to Möbius, he makes a sharp distinction between congenital cases and those which develop after birth, and, as the anatomical basis for the congenital cases, he assumes a lack of development somewhere in the motor path from the brain to the muscles.

Last year Schmidt⁷ reported a case in which there was an absence of the left pectoral major, combined with defects in the muscles supplied by the XII., VII. and VI. cerebral nerves.

⁴ Schultze: *Neurolog. Centralbl.*, xi., 1892, p. 425.

⁵ Bernhardt: *Ibid.*, xiii., 1894, p. 2.

⁶ Kunn: *Beiträge zur Augenheilkunde*, Heft xix., 1895, p. 1.

⁷ Schmidt: *Deutsche Zeitschrift. f. Nervenheilk.*, x., 1897, p. 400.

From this short glance at the literature, it may be stated that congenital abnormalities of the muscles occur not very infrequently; that they are most common in the pectoral muscles and the muscles of the eyes, and that the facial muscles are at times implicated, usually in association with those of the eyes, but at times alone.

The facial defect, when it has been combined with that of other muscles, has been bilateral; when it has been uncomplicated, it has been unilateral.

As far as I have been able to discover, a satisfactory case of congenital facial diplegia has not, or, rather, had not been reported until I showed one of my cases to the Johns Hopkins Hospital Medical Society. The two cases which I have observed have an added interest, as they are brothers.

Seth O., æt. 21; Basher O., æt. 19.

The parents of these two boys are strong, healthy country people. There is no history of alcoholism and no reason to suspect syphilis. The mother's aunt gave birth to a child with only one arm, and the mother herself had a baby with a deformed foot. This child lived only two hours. Other than this there is no history in the family of congenital deformities. The mother accounts for the condition of her boys by "maternal impression", as she had been very much affected, while pregnant with the first boy, by hearing of a minister in her church who was unable to move any of the muscles of his face. Before the birth of the second case she was very anxious for fear that this child would be born also with a deformed face. It may be remarked in passing, that her anxiety about the faces of the subsequent children, of whom there were three, all boys, had no effect upon the development of their facial muscles. An elder daughter is also perfectly normal. Three other children died in infancy.

Case I.—Seth O., æt. 21. The patient, a second child, was born without instruments, although the labor was difficult and protracted. His mother noticed soon after his birth, that his under lip drooped, and that he did not close his eyes when sleeping. He was able to suck without difficulty by the use of his tongue. When he was old enough to smile, his face remained expressionless. The patient learned to walk at thirteen months, and to talk when about two years old, although he has never learned to pronounce certain words distinctly.

Dentition began very early, the first tooth being cut when he was little more than six weeks old. He developed normally, learned to run and play games as other boys, and except for his expressionless face there was nothing to be noticed about him. He left school when he was twelve, as he then became sensitive as to his appearance. As a child he was ill with measles, and his mother thinks that his deafness is a re-



FIG. I. (Case I.)

sult of this disease, although he had no discharge from his ears, or any symptoms referable to it.

The examination (Nov. 3d, 1896) at his home in a neighboring State, shows him to be in general a well-developed young man, rather shorter than the average (Fig. I.) His intelligence seems to be good, his speech is somewhat in-

distinct, as the labials cannot be given their proper sounds. B is pronounced D, F ech, M is N, V is ch or ge, the sound of W is poorly given. His face is perfectly expressionless, forehead smooth, without wrinkles, eyes wide open, mouth open, lower lip large and everted. The lobe of the left ear is misformed, there being a distinct division between the part next to the cheek and the rest of the ear. On the right side there is some indication of this abnormality. His teeth are



FIG. II. (Case 2.) Face in repose.

fairly well formed. The senses of smell and sight show no marked abnormality. His eyeballs are not especially prominent, are straight and freely moveable in all directions, and there is no nystagmus. The pupils are equal and react to light and during accommodation.

The muscles of mastication act well and equally on the two sides. Sensation in the face is normal, and the sense of taste is unaffected.

The patient is entirely unable to raise or to contract his eyebrows. When trying to close his eyes, the eyeballs are rolled up and the upper lids are somewhat relaxed. He is unable to elevate his upper lips or to pucker his mouth, or indeed to close it. On the right side he is able to draw the angle of his mouth somewhat outwards. This motion is impossible on the left side. He can depress and retract the angles



FIG. III. (Case 2.) Face in repose.

The muscles of the soft palate act normally; the pharyngeal reflex is active. He protrudes his tongue in the middle line, and is able to move it freely in all directions. The action of of his mouth by the action of the platysmæ. An electrical examination could not be made. The patient is quite deaf in both ears, being just able to hear a loud-ticking watch upon contact.

his heart is normal in all respects. The muscles of his shoulder girdle, arms, hands, trunk and legs are well developed and of normal strength. The deep reflexes are normal, and no other abnormality of any kind is discovered.

Case II.—Basher O., æt. 19.

Birth normal. The defect in the baby's face was noticed shortly after his birth. He was able to nurse, as was his



FIG. IV. (Case 2.) Showing extent of voluntary control of muscles.

brother, by the use of his tongue. He developed very much as his brother did. When he learned to speak, there was the same difficulty in pronouncing words, etc. He cut his teeth at the usual time. He was an active boy, fond of all out-door games. The facial defect never changed from his earliest infancy. He has learned to smoke, holding the cigarette in

his tongue, and, indeed, he has taught his tongue to do many of the services that are usually performed by the lips.

Present condition. Nov., 1896. Figs. II. to V.

The patient is a well-developed youth. His intelligence is good. His speech shows the defect noted in the case of his brother, that is, he is unable to pronounce the labials. His face is remarkably expressionless, eyes are wide open and



FIG. V. (Case 2.) Faradic stimulation of platysma.

prominent, mouth is held open, lower lip is large and pendulous. The lower jaw is protruded, the lower teeth being more than half an inch beyond the upper teeth. His teeth are poorly developed. The lobe of the right ear is notched, in a similar manner to that of his brother.

The patient's vision is normal, his eyes are in the median

line, freely moveable in all directions, and there is no nystagmus. The pupils are equal and react to light and during accommodation. The muscles of mastication are unaffected, the sensation of the face is normal. Taste is acute on the fore part of the tongue. He is unable to elevate or contract his forehead in the least. When endeavoring to close his eyes, the eyeballs are rolled upward and the lower lids are relaxed (Fig. IV.). He cannot elevate his upper lip, but is able to retract and depress the corners of his mouth by the use of the platysma. The buccinators have also retained some power. Electrical stimulation from the root of the facial nerve causes contraction in the muscles which can be voluntarily moved, and in those moving the ear. By direct stimulation the platysma can be brought into play (Fig. V.). It takes relatively strong current to produce these contractions. The patient is deaf in both ears, and can hear a loud-ticking watch only when it is within two inches of his ears. The movements of the soft palate and tongue are normal. The pharyngeal reflexes are normal. The development in the arms, trunk and legs is excellent. Nothing else abnormal is noticed.

The patient entered the hospital in Dr. Halsted's wards and underwent two plastic operations devised to bring his lips closer together. In the first operation a bit of lower jaw was excised, and in the second the redundant portion of the lip was cut away. There was marked improvement, his lips being nearly approximated.

During the second operation it was noticed that there was very little muscular tissue in the lip, it being composed largely of fat. Fibres of what was supposed to be the platysma were made out.

That the muscle defect in these cases is a congenital one, I think cannot be doubted. In the first place, the mother is quite sure that the defect was present when the children were born, although she cannot state that she noticed the deformity immediately after birth. The fact that the malformation occurred in two members of the same family, and that it is bilateral, speaks for its congenital origin, and the occurrence of other faults in development, the misshaped ears, and, possibly, the deafness, lends added weight to this view. The character of the paralysis itself is quite similar to that which was found in the other congenital cases.

There are 14 cases of facial paralysis combined with

eye-muscle defect; these have been reported by Graefe,⁸ 1880; Harlan,⁹ 1881; Chisolm,¹⁰ 1882; Armaignac,¹¹ 1886; Möbius,¹² 1888; Schapringer,¹³ 1889; Bernhardt,¹⁴ 1890; Bloch,¹⁵ 1891; Fryer,¹⁶ 1892; Bach,¹⁷ 1893; Remak,¹⁸ 1894; Gesèpy,¹⁹ 1894; Schmidt (*loc. cit.*), and Procopovici,²⁰ 1896.

I have not included Rechin's two cases, because in the one the onset is said to have been in the fourth year, and in the other there was no true facial paralysis, nor Hanke's case, as the history of the onset is not satisfactory.

In these 14 cases, the defect in the VII. nerve was combined with that in the VI. no less than 12 times, and in 7 cases there were no other cranial nerves involved. In one, the sensory portion of the V. was also involved, and in another there was a partial defect of the motor V. and of the XII., and in 2 cases the external ophthalmoplegia was practically complete, involving the III. and IV. nerves as well as the VI. The fifth case is the interesting one reported by Schmidt, in which, combined with the pa-

⁸ Graefe: Case 4, *Handbuch d. ges. Augenheilk.*, 1880, vol. vi., p. 60, *cit. from Kunn.*

⁹ Harlan: Case 1, *Trans. of the Am. Ophthal. Soc.*, 1881, p. 216, "Congenital Paralysis of Both Abducens and Both Facial Nerves."

¹⁰ Chisolm: *Archives of Ophthalmology*, vol. xi., 1882, p. 323.

¹¹ Armaignac: Case 3, *Rev. Clin. d'Oculistique*, November, 1886, *cit. from Kunn.*

¹² Möbius: *Münch. med. Wochenschr.*, 1888, No. 667, "Ueber angeborene doppelseitige Abducens Facialislähmung."

¹³ Schapringer: *New York med. Monatschrift*, December, 1889; *Boston Med. and Surg. Jour.*, 1889, p. 635.

¹⁴ Bernhardt: *Neurolog. Centralbl.*, 1890, vol. ix., p. 419, "Ueber angeborene einseitige Trigemini Abducens Facialislähmung."

¹⁵ Bloch: *Berlin Thesis*, 1891.

¹⁶ Fryer: *Ann. Ophthal. u. Otolog.*, Kansas City, 1892, "Case of Congenital, Bilateral, Ext. Ophthal. and Cong. Bilateral Facial Paralysis," *cit. from Kunn.*

¹⁷ Bach: *Centralbl. f. Nervenheilk.*, xvi., 1893, p. 57.

¹⁸ Remak: *Neurolog. Centralbl.*, 1894, No. 7, "Ein Fall von einseitigem angeborenem Defect des Platysma myoides."

¹⁹ Gesèpy: *Arch. d'Ophthal.*, xiv., 1894, No. 5, 273, "Deux Cas d'Ophthalmoplegie Congen. Externe."

²⁰ Procopovici: *Arch. f. Augenheilk.*, xxxiv., p. 34, "Ueber angeborene, beiderseitige Abducens Facialislähmung."

ralysis of the VII. and VI. nerves, there was an unequal paralysis of the XII. pair, the left more than the right, and an absence of the pectoralis major muscle on the left side.

The defect was bilateral in all of these 12 cases, except in Bernhardt's case, in which the paralysis was confined to the right VII., VI. and V. nerves. Bernhardt believed that the condition was caused by some injury to the nerves at the base of the brain, which they had received during birth, and it is doubtful whether the case should be included with the others. I shall have to return to this case, in speaking of the pathology of the condition.

The two cases in which the VI. nerve was normal are of particular interest. The first is that of Armaignac, in which there was a left-sided defect of the orbicularis palp., and probably also of the frontalis, combined with paralysis of the levator palp. and the superior rectus on the same side. Remak's case is the second. In this case the left platysma, including the quadratus and triangularis menti, was absent or paralyzed, and there was also a bilateral defect in the levator and the superior rectus.

As a rule, all the muscles supplied by the VII. were not affected. In Schmidt's case there was complete paralysis. The notes in Fryer's, Bernhardt's, Schap-ringer's and Bach's cases are not specific on this point. Therefore, in the 10 cases in which there was a definite note, the paralysis was incomplete in 9. The muscles which draw the mouth outwards and downwards (the platysma, etc.) are particularly likely to be spared. They were spared alone four times; once the orbicularis palp. was also not affected, and in two other cases the paralysis was confined to the upper branch of the VII. In Bloch's case this paralysis was of the lower branch of the VII. In Remak's patient the platysma, the quadratus menti, and the triangularis menti were the only muscles paralyzed, involving just the muscles which are usually spared.

There are 6 cases in which there was an uncomplicated congenital facial paralysis. These have been reported by Stephan,²¹ Henoch,²² Schultze,²³ Bernhardt,²⁴ Procopovici²⁵ and Bernhardt.²⁶ I have not included Delprat's case (cit. after Möbius), as there is the definite history of the onset at three, after an acute illness; nor Kunn's case, as there is here a very different condition, a right hemiatrophy of all the structures of the face below the eye; bones as well as muscles.

In all of these, except in Procopovici's case, the defects were unilateral, three times on the left and twice on the right side. I shall speak of Procopovici's case a little further on, as it belongs more to the cases which I have been reporting than to these.

In all but Stephan's and Henoch's cases the paralysis was incomplete, and it was the muscles about the mouth that were spared. In fact, the clinical picture differs in no way, except in being unilateral and uncomplicated, from that of the VII. nerve paralysis in the combined cases. Bernhardt, in trying to answer the question as to whether these cases should be considered as strictly congenital, shows that they differ in no way from those which are caused by injury to the VII. nerve. That all the muscles supplied by the VII. nerve are not paralyzed, and that there is no secondary contraction, does not, as Kunn thought, serve to distinguish them.

Facial paralysis due to the injury of the nerve at birth is not a very uncommon accident; it is usually caused by the application of forceps, but may occur even during

²¹ Stephan: *Rev. de Med.*, July, 1888, p. 548, and *Nederl. Tijdschrift*, 1888, p. 113, cit. from Bernhardt.

²² Henoch: *Vorlesungen über Kinderh.*, 1897, ix. Auflage. Also abst. Bernhardt, *Neurolog. Centralbl.*, ix., p. 423.

²³ Schultze: *Neurolog. Centralbl.*, xi., 1892, No. 14, p. 425.

²⁴ Bernhardt: *Neurolog. Centralbl.*, 1894, xiii., p. 2.

²⁵ Procopovici: *Arch. f. Augenheilk.*, 1896, xxxiv., p. 44.

²⁶ Bernhardt: *Neurolog. Centralbl.*, xvi., 1897, p. 296.

normal labor. ²⁷ ²⁸ This paralysis is, in the great majority of cases, a transient one, and the recovery is quite complete. In some cases, however, the paralysis does not get well, and the condition persists throughout life. When this is the case, the symptoms are identical with those described in the five cases under discussion.

As an example of this, I may give briefly the history of a case that I have had under observation for some time. The patient is a young man of excellent health, who, except for his facial paralysis, shows no abnormalities. He has been told by his parents that the condition was noticed directly after birth, and that his birth was very protracted and difficult. He thinks forceps were used, although of this he is not certain. He is certain, however, that the paralysis has always been ascribed to injuries received to his face during birth, and that the condition has not changed since he can remember. At present the left side of his face is almost completely paralyzed. The forehead cannot be raised nor the eye closed, nor the upper lip elevated, and the lips cannot be puckered. He can, however, draw the left angle of his mouth outwards, throwing the cheek into longitudinal folds. He is not able to contract voluntarily the platysma on either side. Electrical stimulation of the left VII. nerve causes contractions of the muscles, moving the angle of the mouth outwards, and nothing else. Another patient, a woman, 34 years old, who has a left-sided facial paralysis, gives the history of having had it since her birth, which was non-instrumental; but, as I have been unable to confirm the history, I shall simply mention it in passing.

Bernhardt, with great fairness, concludes that, although the occurrence of an isolated, unilateral, congenital facial paralysis, or, perhaps, better, an incomplete development of the nerves and muscles in the distribution

²⁷ Geyl: *Centralbl. f. Gynäkologie*, xx., 1896, p. 634.

²⁸ Knapp: *Ibid.*, xx., 1896, p. 705.

of the facial nerve on one side, cannot be denied, still, its occurrence has, as yet, not been definitely demonstrated.

Procopovici (loc. cit. p. 45) refers briefly to an interesting case, which he says had lately come under observation. It is that of a man, 18 years old, in whom there had been, since his birth, a paralysis of the upper branch of the facial nerve. All the muscles which are supplied by the facial nerve were active, except only the orbicularis oculi and the frontalis, which were paralyzed on both sides in almost equal intensity. In other respects the patient was well. (This is the full note.)

Procopovici refers also to another case in the footnote on page 44. A woman, aged 34, had been born with a paralysis, which was more intense on the right side. It affected the muscles of the forehead, the orbicularis palpebrarum and all the muscles of the face. The muscles of the soft palate were not affected. Meynert thought, on account of the distribution, that it was a peripheral paralysis of all the branches of the facial nerve, external to the Fallopian canal. Accidents during birth were to be excluded. The abducens was normal, and hearing was unaffected.

These are the only two cases that I have been able to find of uncomplicated bilateral congenital facial paralysis, and in these cases the histories are so meagre that it is difficult, or impossible, to definitely determine their character. The first of these cases appears to me to be particularly interesting, as the paralysis involves the upper branch of the facial nerve, the branch which is believed by some to arise near the nucleus of the third nerve. In this connection I shall recall Armaignac's case, in which there was a congenital defect of the orbicularis palpebrarum and of the frontalis of the left side, combined with a similar defect in the levator palpebræ and rectus superioris of the same side. In Gesèpy's case, in which there was a bilateral paralysis of the orbicularis palpebrarum, there was complete external ophthalmoplegia.

As for the pathological basis which underlies this interesting condition, very little can be definitely said. Its pathology is almost entirely speculative. Möbius, who was the first to make any exhaustive study of these cases, associated the cases of congenital paralysis of the facial and abducens nerves with the other cases of congenital paralysis of the eye muscles, and he brought these congenital cases into relation with cases which developed in infancy and childhood, and presented symptoms which were quite similar. That the congenital cases might be due to some defect in development, an aplasia of the motor apparatus, Möbius recognized, but he thought it was better to assume the same process for both the congenital and acquired cases, and this process he believed to be an atrophy of the nuclei from which the nerves arise.

Kunn, in his monograph, written several years after Möbius' second paper, reviews the whole literature, and makes a sharp distinction between the congenital and the acquired cases. He bases this distinction upon what he considers definite clinical differences; these are in relation to the paralysis of the ocular muscles, and do not particularly concern us at this time. He believes that the congenital cases are not due to an atrophy of the nuclei, but should be considered as a defect in the development in the motor mechanism, and he announces the theory that the defect may be anywhere in the motor path, from the cortex of the brain to the muscles. Kunn admits that this theory is based on a very slight anatomical foundation.

In congenital defects of the eye muscles, which have been operated upon, the muscles have at times been found wanting, and at times in every degree of development, up to what appeared perfectly normal muscles. In certain cases microscopic examination of a bit of excised muscle showed a condition quite similar to that which is described in progressive muscular dystrophy. In regard to the central nervous system, there are really no ex-

aminations that speak definitely as to the condition of the nuclei in these cases. Bernhardt examined the brain in a case in which the right VII. nerve, the VI. nerve and the sensory portion of the V., on the same side, were paralyzed. The defect was noticed shortly after birth. The child died when it was nine months old. Two foci of softening were found—one superficial in the right side of the pons, the other more extensive in the right corpora quadrigemina. The nuclei of the cerebral nerves were said to be normal. The peripheral nerves were not examined. Bernhardt himself believes that there was an injury to these nerves at the base of the brain during birth, and it is very doubtful whether this case should be included with the strictly congenital cases. Siemerling²⁹ describes the autopsy on a man who had had a congenital ptosis of the left eye, and who died from general paresis. He found a degeneration in certain cells of the nucleus of the third nerve. The degeneration was bilateral, and suggested rather a later process than one which had been in existence for fifty years. The character of the process, and the fact that the patient had general paresis, and that the lesion was bilateral, whereas the muscle defect was unilateral, would seem to justify Kunn's objection to considering this a conclusive case.

But it would seem to me that Kunn was probably right in distinguishing the congenital cases from the acquired cases, and, from the point of view of congenital facial paralysis, it is interesting to note that Möbius was unable to find a case of abducens-facial paralysis which developed in childhood. The case which he referred to as a doubtful case, that of John Thompson,³⁰ seems more likely to have been due to a neoplasm of the medulla than to a nuclear atrophy.

That congenital facial paralysis is almost always as-

²⁹Siemerling: *Arch. f. Psych.*, xxiii., 1892, p. 764.

³⁰Edinburgh *Med. Jour.*, vol. xxxvii., 1891, p. 262.

sociated with paralysis of the VI. nerve would lead one to believe that the mal-development is in the medulla, near the origin of these two nerves. That the nuclei of both nerves need not always be affected together is shown by the occurrence of uncomplicated congenital VI. nerve paralysis, and there seems no reason for assuming that such a condition could not occur in the nucleus of the VII. nerve, and that is what I assume has happened in the boys whose cases I have reported. If, in fact, a mal-development, or perhaps a non-development, of the nuclei accounts for the occurrence of these cases, it does not follow that the muscles themselves must be absent. The cases which Frl. v. Leonowa³¹ reported demonstrate that muscles may develop independently of the central nervous system. She examined two monsters which were entirely without brain or spinal cord. The dorsal root-ganglia and nerves growing from them had developed, as had the muscles. In many cases of congenital ptosis curious associated movements occur in relation to movements of the jaw, and this would also indicate that the defect was in the nucleus, and that this associated movement had its representation apart from the nucleus of the third nerve, probably in the nucleus of the V. nerve.

That there may be a congenital absence of the muscles, the cases of the absence of the pectoral and other muscles seem to prove, but it is hard to understand why such a defect should be strictly limited, as it was in our cases, to the distribution of one nerve, if the nervous mechanism had nothing to do in determining it, and when we associate with this, as so often happens, a defect in just that one of the six external muscles of the eye whose nucleus lies in close relation to the VII. nucleus, any other explanation than a defect of these nuclei seems very far-fetched.

Schmidt's case, in which there was a defect in the VI.

³¹ v. Leonowa: *Neurolog. Centralbl.*, 1893, pp. 218 and 262; 1894, p. 729.

and VII. nerves, combined with absence of the left pectoralis major muscle, does not demonstrate that the process was the same in the muscles of the face and the muscle of the chest. Congenital abnormalities are rarely unassociated with other malformations, and even if we assume a different cause for the absence of the pectoralis major and paralysis of the facial muscles, it would not be surprising that they both might occur in the same individual. We do not, however, know the cause of the congenital absence of individual muscles, and it may depend upon some fault in the development of the central nervous system.

There is no proof that a congenital abnormality in the upper motor segment could produce defects of this character, and I agree with Möbius that such a lesion is not to be assumed. In conclusion, we must acknowledge that we do not know the anatomical basis for the cases of congenital facial paralysis, but it is more in accordance with the known facts to assume some fault in the development of the nucleus of the VII. nerve to account for these cases.

FACIAL PARALYSIS COMBINED WITH EYE-MUSCLE DEFECT.

Graefe: Handb. d. ges. Augenheilk., 1880, vol. vi., p. 60.

Cit. from Möbius. Left VII. nerve paralyzed. Right upper and middle branches weak. Bilateral VI. nerve paralyzed. Smell and taste somewhat affected.

Harlan: Trans. of the American Ophthal. Soc., 1881, p. 216. Bilateral paralysis of VII. and VI. nerves (complete?). Taste normal. The platysma active. Slight downward movement of mouth.

Armaignac: Révue clin. d'Oculistique. November, 1886.

Cit. from Kunn. Left paralysis of orbicularis palp. (frontalis probably), and levator palpebræ and rectus superioris.

Chisolm: Archives of Ophthalmology, vol. xi., 1882, p. 323. Bilateral paralysis of VII. and VI. nerves. Muscles about lower lip retaining some power.

- Möbius: Münch. med. Wochenschr., 1888, p. 667. Bilateral paralysis of VII. and VI. nerves. Some retention of muscles about mouth. Left side moved a little out while talking. Elect. m. m. which draw mouth out and down L. and R. are excitable. Also buccinators. Smell, hearing and taste normal.
- Schrapinger: Boston Med. and Surgical Journal, 1889, p. 635. Bilateral paralysis of VII. and VI. nerves. Paralysis of motor V. and XII. nerves. VII. nerve paralysis not specified as to extent. Other congenital abnormalities.
- Bernhardt: Neurolog. Centralbl., 1890, vol. ix., p. 419. Paralysis of right VII. and VI. nerves, also sensory disturbance. Infant, autopsy.
- Rechin: Klin. Monatsbl. für Augenheilk., 1891, p. 340. Cit. from Kunn. Two cases, doubtful. First said to have developed at four years. Bilateral paralysis of VII., especially lower branch. Nearly complete ophthal. external. Second case, facial muscles thin, badly developed, but not paralyzed. React normally to electricity. Ophthalmoplegia ext. All muscles of body badly developed.
- Bloch: Berlin Thesis, 1891. Case 28. Boy nine months old. Bilateral paralysis of VI. nerve. Bilat. paralysis of VII. nerve, lower branch. Bilat. club-foot and other abnormalities. The affection was congenital. (Note short.)
- Fryer: Ann. Ophthal. u. Otolog., Kansas City, 1892. Cit. from Kunn. Bilateral paralysis of VII. and VI. nerves.
- Bach: Centralbl. f. Nervenheilk., xvi., p. 57, 1893. Man 27. Bilateral ptosis. Bilat. ophthal. ext. Condition congenital. Facial muscles were flaccid. Frontalis and corrugator contracted. Eyes can be closed. Features expressionless. Lips moved only slightly during speech; no absolute paralysis. Operation on ptosis. Muscle normally placed. No noticeable abnormality.
- Rémak: Neurolog. Centralbl., 1894., No. 7. Paralysis of

- left VII. nerve, and bilateral paralysis of levator and superior rect. The platysma, the quadratus and triangularis menti were the only facial muscles affected.
- Gasèpy: Arch. d'Ophthal., xiv., 1894, No. 5, p. 273. Cit. from Kunn. Bilateral paralysis of VII. nerve (orbic. palp.) and ophthal. ext. nearly complete.
- Hanke: Wiener klin. Wochenschr., 1894, 46. Abstr. Schmidt's Jahrbücher, vol. 246, p. 22. Doubtful case. History of onset unsatisfactory. The "staring look" had always existed. Woman, 26. L. ptosis 7 years previously. Examination: Facial muscles flaccid. Muscles about mouth and eyes less exc. to elect. Ophthal. ext.
- Procopovici: Arch. f. Augenheilk., xxxiv., 1896, p. 34. Bilateral paralysis of VII. and VI. nerve. Muscles about angle of mouth retained and the orbicularis palp.
- Schmidt: Deutsche Zeitschr. f. Nervenheilk., x., 1897, p. 400. Paralysis bilateral of VII. and VI., also of XII. L > R. Also absence of left pectoral major. The VII. nerve paralysis was complete.

UNCOMPLICATED CONGENITAL FACIAL PARALYSIS.

- Delprat: Weekb. v. het Nederlandsch. Tijdschr. vor. Geneek., November 29th, 1890, No. 22, p. 697. Cit. from Möbius. Not accepted by Möbius, but included by Kunn. Onset said to have been after an acute illness when 3 years old. Examined when 16. Right, weakness of muscles which elevate mouth, especially zygoid maj. Left, weakness of all muscles except corrugator supercillii, orbicularis palp. and the zygoid. Decreased elec. excitability. Left amblyopia.
- Henoch: Vorlesungen über Kinderheilk., 1897, ix. Auflage, p. 22. Boy, 10. Left VII. nerve paralysis, which had existed since birth; also paralysis of soft palate and deafness on left side. No condition in ear to account for deafness.
- Stephan: Révue de Med., 1888, July, p. 548. Abst. Bernhardt, Neurolog. Centralbl., ix., p. 423. Noticed soon

after birth. Examined when 32. Left VII. nerve paralyzed. Soft palate paralyzed. M. M. not excitable to elec. Taste normal. Deaf in left ear.

Schultze: *Neurolog. Centralbl.*, xi., 1892, No. 14, p. 425. Noticed directly after birth. Examined when 4. Total left VII. nerve paralyzed. Pupils L > R. Slight nystagmus in lateral position. Elec. examination, strong current causes contraction in left orbicularis oris, and nothing else on left side. This muscle is not noted as acting normally. No other abnormality.

Bernhardt: *Neurolog. Centralbl.*, 1894, xiii., p. 2. Noticed 2 weeks after birth. Examined when 24. R. VII. nerve paralyzed (forehead, eye and upper lip). Muscles of under lip and chin retained. Only the active muscles respond to elec. from nerve. Slight nystagmus in lateral position. Taste normal.

Kunn: *Beiträge zur Augenheilk.*, Heft xix., 1895, p. 1. Case doubtful. Noticed just after birth. Examined at 16. Abnormal development of right side of face. Eyes and forehead normal. Below, marked asymmetry of face. Masseters and bones less developed. Weakness but no actual paralysis of VII. and V. nerves. Normal electrical response. Taste normal.

Bernhardt: *Neurolog. Centralbl.*, xvi., 1897, p. 296. Noticed in first days of life. Examined when 7. Face, R. VII. nerve paralysis. Muscles of lower lid acted, and she could pucker her lips as in whistling. Elect. examination: Marked decreased excitability in orbic. oris to direct current. Is excitable from other side. Nothing from nerve. Taste could not be tested.

Procopovici: *Arch. f. Augenheilk.*, xxxiv., 1896, p. 34. Case I. Since birth. Examined at 18. Bilateral paralysis of orbic. oculi and frontalis. No other abnormality. Case II. Noticed soon after birth. Examined at 34. Bilateral weakness of VII. nerve, R. more than L. Eye muscles normal. Hearing normal.

DISCUSSION.

Dr. Wm. G. Spiller referred to the relationship between the condition described by Dr. Thomas and progressive muscular dystrophy of the Landouzy-Dejerine type. In the latter affection the muscles about the eyes and mouth are much involved, and the disease may exist for years with the atrophy limited to the muscles of the face. In the patients whom Dr. Spiller had seen, one might easily believe, at first sight, that the seventh nerves were affected. The existence of the disease in Dr. Thomas' cases, in two members of the same family, was suggestive of progressive muscular dystrophy, though the speaker did not intend to make a diagnosis different from that formed by Dr. Thomas.

Dr. B. Sachs said that while listening to Dr. Thomas' paper, it occurred to him that the cases reported were possibly instances of progressive muscular dystrophy of the Landouzy-Dejerine type. If the condition could be traced back to birth, however, that would militate against the latter diagnosis. In one of the photographs shown by Dr. Thomas it appeared that the eyelids could be very nearly closed, if not completely so, which would lead one to entertain the idea that the muscular element was very much more at fault than the neural. The speaker said he was inclined to believe that in these various forms of congenital defective development, the defect was not observed merely in the neural part of the motor tract, but that other parts of the body were also poorly developed. In one case which he had reported, a defect of the pectoralis major muscle was found in connection with a distinct defect of the scapula. It was probable, he thought, that these cases were not necessarily neural in origin; while some of them might be, others showed that the muscular or osseous system alone was involved.

Dr. Hugh T. Patrick inquired whether an electrical examination of the eighth nerve was made?

Dr. B. Onuf asked whether Dr. Thomas attributed any etiological importance to maternal impression in these cases.

Dr. Thomas, in closing, said he did not believe that maternal impression had anything to do with the disease in the cases he had reported. It might be of interest, however, to state that in one case in the literature where a child was born with a facial defect, the mother, during her pregnancy, had made frequent visits to a physician, who was treating her mother with electricity for a facial paralysis.

Dr. Thomas said that the question of a probable relationship between this condition and progressive muscular dystrophy had been brought up before, and while the anatomical

changes in the muscles in certain cases of congenital ptosis were quite similar to those found in this disease, this did not, by any means, demonstrate that the cause of the atrophy was the same. In his cases the defects of development were not confined to the muscles of the face; there was also a defect of the ear and a defect in the hearing, and on this account he regarded the symptoms as congenital. In reply to Dr. Patrick, the speaker said he did not make an electrical test of the eighth nerve. In conclusion he expressed the opinion that it would be unusual to see a case of muscular dystrophy of the facial type which dated from birth and persisted until the twentieth year, in which only the muscles of the face were affected.

190. LA MALADIE DU SOMMEIL, ET SON BACILLE (The Sleeping Sickness and its Bacillus). Cagigal and Lepierre (*La Médecine Moderne*, 9, 1898, p. 60).

Cagigal and Lepierre, the authors, observed a negro, 16 years of age, a native of Angola, who suffered with the sleeping sickness for three years. He was in the hospital of Coimbre more than two months, during which time there was an elevation of temperature, with remissions, up to some weeks before death, but during this latter period the temperature was constantly subnormal. The only physical peculiarity further was constant passage of ammoniacal urine with an excess of phosphates and relative azoturia. The examination of the blood showed a constant presence of bacilli and spores. The cultures of blood from the arm and hand on serum, gélose, gelatine, bouillon and peptone resulted as follows: At moderate temperatures most of the media remained sterile, except those of serum and gelatine, which produced homogeneous colonies, the serum after three days, the gelatine only after four weeks. With the culture upon serum the same results were obtained as with the blood. There was some difficulty in development of the microbe upon the culture media except with serum, a point upon which the authors insist. The microbe observed in the blood and cultures is a straight bacillus, sometimes incurved, a little larger at the extremities than at the centre; it makes filaments, it is very little mobile, takes anilin stains well, does not color with Gram, and appears not to have any processes; free spores were observed both in the cultures and in the interior of the microbes. The general aspect of the preparations resembles those of the anthrax bacillus; the growth is best at a temperature of from 30 to 37 degrees C.; a moist heat of 75 degrees C. kills it in a minute; it is a true aërobic bacillus.

Postmortem—Some microbes were found in the intraperitoneal fluid. Inoculations of rabbits and guinea pigs produced certain effects not unlike those observed in the human subject—identical temperature-curves, sub-normal temperature preceding death, loss of weight, an appearance of depression, feebleness, especially of the hind quarters, ammoniacal urine, and death in from twenty-five to fifty days.

MITCHELL.

A CASE OF MULTIPLE SYPHILITIC NEURITIS.¹

By FRANK R. FRY, A. M., M. D.,

St. Louis.

The subjoined history will, perhaps, be traced more satisfactorily if first epitomized as follows: Male, 32 years of age. March, 1897, a rather severe headache, which yielded to antisyphilitic treatment within about ten days. June, a syphilitic lesion of the tonsil. July and August, headache again, but not severe; no symptoms of focal disease of the nervous system. October 26th, a sudden right hemiplegia, involving the arm and leg, but not the face; probably due to a lesion at the internal capsule; no other lesions of the central nervous system. November 15th, beginning of a paraplegia with hyperæsthesia and anæsthesia of all the extremities, especially of the hands and feet, and later a complete paraplegia, with flaccid muscles and reaction of degeneration in the legs, and absent knee-jerk, and marked weakness of the hands and forearms. A diagnosis of multiple neuritis, involving all of the extremities. March, 1898, returning motility, and later a gradual recovery from paraplegia, with returning knee-jerk.

In particular, the history is as follows:

E. A. W., aged 32 years; a large, well-built man; German parentage; family history unimportant; no previous illness of importance; good habits; never has used tobacco or alcohol excessively; city salesman for a wholesale queensware house.

He first consulted me March 13th, 1897, complaining of

¹Read at the twenty-fourth annual meeting of the American Neurological Association, May, 1898.

headache and lassitude. I concluded that he was suffering from a grippe cephalæa, and prescribed accordingly. He called several times within a week. The treatment afforded no relief. March 22d he complained a great deal of his head. In the course of a careful examination I found the temporal ridges and shins slightly tender to pressure. On antisyphilitic treatment, the headache entirely disappeared within about ten days, and his general condition was much improved. A careful inquiry and physical examination at this time failed to elicit a history or signs of syphilis, save the periosteal tenderness just mentioned. Subsequently, however, I obtained from him data which pointed quite conclusively to an infection in September of 1896. I tried to impress him with the necessity of keeping himself under observation and treatment, but after two weeks, being very much occupied, he discontinued his visits.

June 22d he returned, complaining of a sore throat. He stated that he had discontinued all treatment a month after his last visit, having felt entirely well. I found a large excavated ulcer on the left tonsil, and the glands at the angle of the jaw swollen and painful. He was examined by Dr. W. C. Glasgow, who pronounced the condition syphilitic. After this I saw him at intervals of a few days to two weeks, and treatment was continued with fair regularity all summer. The lesions in the throat and neck disappeared slowly.

My notes show that on July 25th and August 13th he was complaining a good deal of headache, and that I made careful examinations without finding evidences of disease of the nervous system.

October 24th, in my absence from the city, he came to the office and was examined by Dr. M. A. Bliss, who found him with a right hemiplegia. He had retired the evening before with a numb, heavy feeling in the neck, shoulder and arm, and awoke in the morning with the hemiplegia and some slight headache and dizziness. He stated that he had been feeling the paræsthesiæ in shoulder and arm for some days prior to the stroke, and had ascribed the condition to rheumatism.

He was admitted to the Missouri Baptist Sanitarium October 26th, and remained there until February 13th, 1898—111 days. During this time I saw him almost daily, and kept full notes of the case. October 26th—the day after admission—I made a general examination. Patient walked with a hemiplegic gait, and the grasp of the right hand was feeble. He had a partial right hemiplegia, involving the leg most, the hand less, and the face and tongue not at all. He could ascend the stairs unassisted, and had fair use of his hand, for example, in dressing and feeding himself. The knee-jerk and wrist-jerk

on the right side were exaggerated. The sensorium was clear. He still complained of slight vertigo, especially on bending over or on suddenly rising from a seat. He also complained of paræsthesiæ in the arm and hand, numbness and heaviness. The expulsive force of the bladder was somewhat weakened. In this connection I will state that the condition of the bladder remained practically the same through the course of his illness. It was never necessary to use a catheter. None of the cranial nerves were involved, and there was no evidence at this time of more than one lesion of the nervous system. It seemed probable that this was a gumma, and that it was located at the internal capsule. Dr. L. Bremer saw the patient a few days after he entered the sanitarium, and concurred in our opinion of the probable nature and location of the lesion.

Active antisiphilitic treatment was continued. At the end of two weeks there was considerable improvement in the general condition of the patient and that of the paretic members, yet I felt that he was not responding to treatment as well as could be desired.

November 15th to 20th he began to make frequent complaints of numbness and weakness in his feet and legs. More careful observations on the 20th showed that there was some hyperæsthesia, which was greater in the left foot and leg. Within a few days the left foot and leg were as paretic as the right. By December 6th he was unable to walk, and took to a roller chair; meantime, he was complaining of an increasing tingling, numbness and weakness in the hands.

December 10th his condition was as follows: A symmetrical paraplegia; he could not walk, but could get into his chair with the assistance of an attendant. The muscles were flabby and slightly tender to pressure. Both knee-jerks were gone. He complained of an unpleasant degree of hyperæsthesia of both hands and feet and legs and forearm, and mostly of those of the left side. The grasp on both hands was enfeebled.

During the following month there was little change in his condition. Mild galvanism was applied daily by Dr. M. W. Hoge, and it was noticed that in the muscles of the legs especially there was a qualitative change in the reaction, the A. C. C. being equal to or greater than the C. C. C.

January 10th (1898), he seemed to be getting stronger in his legs. On January 18th he first undertook to walk with the assistance of two attendants, and from that date gained rapidly, so that at the end of two weeks he was moving about his room with the support of a walking-stick. The gait was not spastic; there was a distinct toe-drop, the so-called steppage gait.

He has been constantly under observation since leaving

the sanitarium, February 13th. Improvement has steadily continued. March 29th, returning knee-jerks were first demonstrated; the right quite feeble without re-enforcement, the left only showing with re-enforcement.

April 4th, 10th, 18th, 25th, he still complained of slight numbness of his fingers and toes, and of considerable sensitiveness of the plantar and palmar surfaces, and some general slight soreness and stiffness of the muscles of the extremities. The æsthesiometer showed only slight obtundity of tactile sense.

May 27th, an examination showed the following: The pupils were equal and reacted normally. The face was symmetrical; no evidence of involvement of any of the cranial nerves. On a stiff dynamometer the right hand registered 170, the left 155. He had a powerful grip. The right knee-jerk was considerably stronger than the left, the latter only coming out well on re-enforcement. Station was good, and there was no incoördination in any of his movements. He could alight from a street car before it came to a full stop. He could walk twenty blocks without much fatigue, and averaged about two hours a day on his feet about his place of business, but was conscious of not having regained his normal strength and endurance. The æsthesiometer showed that the tactile sense of the fingers was not quite up to normal, and he still had occasional very slight stiffness and numbness in the left hand. After sitting for some time, he felt some stiffness of the legs, which soon wore away on moving about. Occasionally he had a slight headache in the temples, causing passing annoyance.

The treatment throughout has consisted of inunctions of mercurial ointment, one to two drachms daily, in courses of fifteen to thirty days. Between these courses iodide of sodium was given in increasing doses, the largest reached at any time being 100 grains, *t. i. d.* Strychnine was used almost continuously in doses of 1-15 to 1-30 grain, *t. i. d.*, and mild galvanism a good part of the time.

I believe that the existence of an extensive multiple neuritis in this case will be conceded, and that the manner in which it was engrafted upon the hemiplegia furnishes a very unusual clinical picture for a syphilitic case. Superficially, it bore a resemblance to the so-called syphilitic triplegia, *i. e.*, a paraplegia of spinal origin following upon a hemiplegia of a cerebral origin. But I think I have given an extended enough history of the case to show that it could not have been mistaken for this rather fa-

miliar condition, or for a diplegia due to extensive cerebral syphilis.

I have assumed that the neuritis was of syphilitic origin, not simply because the patient was syphilitic and the disease rather intractable to treatment, but because there seemed to be no other probable explanation of it. There was nothing in his occupation or habits which predisposed him to it. He had never shown rheumatic or lithæmic tendencies, and had no renal trouble. Mercury had been used before the neuritis appeared, but it was not discontinued. On the contrary, it was used more freely after the neuritis was manifest than before its appearance. There were at no time the evidences of a mercurial cachexia. In January for a few days the teeth were a little tender when jarred together, and this was the nearest approach to the full physiologic effect of the drug observed at any time, although constant watch for this effect was kept. A diffuse polyneuritis from mercury is a very rare occurrence, and a condition of the kind without considerable pain and local lesions in the joints and muscles and an apparent cachexia would be still more rare.

The rarity of an extensive peripheral neuritis due to syphilis is noticed by all authorities, and generally they call attention to the distinction which should be made between a neuritis proper and a postsyphilitic degeneration of peripheral nerves. The necessity of this distinction from a pathological and clinical standpoint is evident enough. Usually there would not be much difficulty in making it clinically. Yet I can understand that situations may occur where, for a time at least, the symptoms may be confusing.

It will be noticed in this case that the syphilis was about one year old at the time the neuritis developed.

DISCUSSION.

Dr. Charles L. Dana said that his experience with multiple neuritis and with syphilitic affections of the nervous system

had been such that he had come to regard it as almost a pathological law that multiple neuritis never results from syphilis. Personally, he had never seen a case of that kind, and those which he had seen reported in literature seemed to be open to some other interpretation. One case—that of a patient who had been under his observation for a long time, and which he did not at all regard as one of syphilitic multiple neuritis—was afterwards recorded as such by another physician. In order to prove such a condition, every other possible cause of the neuritis must be excluded. Dr. Dana said he had specimens in his possession from a case where a rather profuse syphilitic exudation into the substance of the cord in the cervical and lower thoracic regions produced symptoms somewhat like those reported by Dr. Fry. In the ordinary cases of multiple neuritis usually much more pain and rather more paralysis are found than seemed to be present in Dr. Fry's case. On the other hand, we know that multiple neuritis is not infrequent, and is sometimes due to causes which we cannot clearly make out. While alcohol is a very common cause, we frequently see cases which must be explained otherwise.

Dr. Sachs said that while he did not care to venture an opinion as to whether Dr. Fry's case was one of syphilitic multiple neuritis or not, he thought Dr. Dana had stated the other side of the question a little too strongly. The speaker was of the opinion that the evidence of a multiple neuritis or peripheral neuritis of syphilitic origin had been fairly well established. Some time ago he had reported the case of a man who had had a number of manifestations of syphilis of the nervous system, among them being ptosis and hemiplegia, and from all of these he had recovered under specific treatment. At one time he had developed a flaccid paraplegia without vesical symptoms, and later a distinct ulnar neuritis, characterized by pain along the distribution of the nerve; at the same time he had mucous patches, and all these symptoms disappeared in the course of a few weeks under proper treatment. Of course, it was possible that this was a mere coincidence, but we have considerable evidence to show that occasionally the peripheral nervous system may be directly diseased as a result of previous syphilitic infection.

Dr. Dana stated that he did not deny the possibility of a syphilitic neuritis of single nerves, but of a multiple neuritis.

Dr. M. Allen Starr agreed with Dr. Dana. He did not think we should confound a peripheral neuritis, due to a gumma, with the typical features we have in mind when we speak of multiple neuritis, which is a clinical entity with well known and characteristic symptoms. Although he had been on the

lookout for a case of multiple neuritis of syphilitic origin during the past ten years, he had never seen one which he was willing to pronounce as distinctly syphilitic.

Dr. J. J. Putnam indorsed the statements of Drs. Dana and Starr. He had never seen a case of multiple neuritis which was clearly of specific origin.

Dr. Leonard Weber said that fifteen years ago he had read a paper before the New York Academy of Medicine, in which he had reported a series of 125 cases of syphilis which had been under his observation more or less constantly for twenty years. Although at that time the clinical features of multiple neuritis were not so well understood as now, he could not recall a single case in that series which presented any of the characteristic symptoms of multiple neuritis.

Dr. Fry, in closing, said Dr. Dana and Dr. Putnam had very justly called attention to the important fact that multiple neuritis is often due to causes which we cannot make out. Therefore, it would be difficult to prove that any case was due to syphilis. In this case it seemed so probable that syphilis was the cause that he had ventured to report it as such.

191. PELLOTIN (Squibb's Ephemeris, Jan., 1897).

This is an alkaloid found in a species of Mexican cactus. It is not soluble in water, but its hydrochlorate is. Dr. Hefter, of Leipsig, observed in himself that after taking five centigrammes (three-quarters of a grain) he seemed very drowsy and ultimately fell asleep. Dr. Jolly, of Berlin, then gave it to a number of patients in the neurological wards of the Charité Hospital in Berlin. The first was a man with alcoholic neuritis, who after an injection of four centigrammes became very drowsy and in a hour later slept four hours. Dr. Hefter observed in himself a diminished pulse-rate, and the same was perceptible in this patient—during the first hour of whose sleep the pulse fell to 56 per minute, rising again to 76 before he awoke. A patient with multiple sclerosis took five centigrammes during the afternoon, and after half an hour he also slept soundly for several hours. Similar results were obtained in other cases. In tabes the soporific action was satisfactory, but the pains returned when the patients awoke. In delirium tremens the effect was less prompt; twelve centigrammes (equal to one and three-quarter grains) were needed to quiet a patient, but no sleep followed. Some patients complained of giddiness and declined to take the medicine, but the greater number did not suffer so. Prof. Jolly says that six centigrammes (equal to about one grain) are equal to one gramme (15½ grains) of trional, or two grains of hydrate of chloral.

FREEMAN

LONG REMISSIONS IN EPILEPSY AND THEIR BEARING ON PROGNOSIS.*

By WHARTON SINKLER, M. D.

The question as to the curability of epilepsy, as well as to what constitutes a cure in this disease, is as far from being settled as it was a century ago. Having chanced to see recently several cases of epilepsy in which there have been long intermissions of the fits, I have been led to look up the subject, and am surprised to find that but few authors have attempted to define what should be considered a cure of epilepsy. In other words, how many years should a patient be free from convulsive seizures before he should be regarded as cured? Neither has any attention been paid to occurrence of long periods of freedom from attacks. The majority of writers on the subject of epilepsy devote their attention merely to the pathology and treatment of the disease. A number of observers report cases in which there has been cessation of the attacks under the influence of certain remedies, and reference is made by some authors, Neimeyer, for example, to the fact that cases are seen in which long intervals between the fits occur independently of treatment. Every writer on the subject of epilepsy asserts that the disease is curable in varying proportions. Some, for instance, declaring "that a large proportion are susceptible of cure," but none pronounce at what period after the cessation of the attacks in an epileptic it is justifiable to consider that the case is cured.

Dana¹ says that five to ten per cent. of epileptics get well, but he does not say what he considers a cure. Gow-

*Read before the American Neurological Association, May, 1898.

¹ Dana: Text Book on Diseases of the Nervous System, p. 411.

ers² says occasionally convulsions occurring in infancy cease at four or five years of age; after twenty years spontaneous cessation does sometimes occur, but it is too rare to be reckoned upon. He does not say how long the fits must be kept away before a cure is established. Osler³ quotes Hypocrites' opinion as holding good at the present day, namely, "that the prognosis in epilepsy is unfavorable when the disease is congenital, or when it endures to manhood, and when it occurs in a grown person without any previous cause the cure may be attempted in young persons, but not in old." Ross⁴ says a few cases of epilepsy are completely cured, but he also fails to say what he means by a cure. Nothnagel⁵ says epilepsy is curable; that spontaneous cures occur in four or five per cent. of the cases, and that some cases are actually cured by treatment, but he does not define his idea of what a cure is, nor does he refer to the length of time which he has known to elapse after an arrest of the convulsions. Hamilton⁶ thinks that some cases of epilepsy are curable, but does not refer to any given period which must elapse before the case may be considered as cured. Niemeyer⁷ says that "in some patients a year, or even several years, may elapse ere a new attack occurs. Recovery must be regarded as rare, and we must beware of building our hopes too sanguinely upon long-continued intermission of fits." Gray⁸ makes a more definite statement in regard to remissions in epilepsy. He says that cases may be free from attacks for ten, fifteen, or even twenty years, and that in his own practice he has known remissions of several years, six or seven. In reference to cure, he says that it is still an undetermined matter as to what constitutes a cure.

² Gowers: *Nervous Diseases*, vol. ii., p. 760.

³ Osler: *Practice of Medicine*, p. 954.

⁴ Ross: *Nervous Diseases*, vol. xi., p. 941.

⁵ Nothnagel: *Ziemssen Encyclopædia*, vol. xiv., p. 277.

⁶ Hamilton: *Pepper's System of Medicine*, vol. v., p. 499.

⁷ Niemeyer: *Practice of Medicine*, vol. xi., p. 361.

⁸ Gray: *System of Nervous Diseases by American Authors*, p. 303.

Starr⁹ has analyzed 167 cases of epilepsy as to the frequency of the attacks, and found that in nine cases the interval was one year and over. Weber¹⁰ records a case of epilepsy in which no fit had occurred for ten years up to the time that the case was reported, and he gives another case in which there was an interval of five years.

I have collected twenty-four cases of idiopathic epilepsy, in which there have been remissions varying from two years to twenty-nine years. These cases have been taken from the case books at the Orthopædic Hospital and Infirmary for Nervous Diseases, and from my own practice, and my friend, Dr. H. P. Boyer, has kindly given me the notes of two cases under his own care, in which the remissions were twelve and thirteen years, respectively. In none of the patients was any surgical operation performed. The length of the remissions is shown in the following table:

Length of remission in twenty-four cases:—Two years, 1 case; from 2 to 3 years, 2 cases; from 3 to 5 years, 9 cases; from 5 to 9 years, 8 cases; 11 years, 1 case; 15 years, 1 case; 21 years, 1 case; 29 years, 1 case.

The case in which the longest remission occurred, that of 29 years, is as follows: A male, aged 38 years, had convulsions while teething, and which continued until he was eight years old. He had then no attack until his thirty-fifth year, when there was a return of the epilepsy, and the convulsions recurred at short intervals until he was seen, three years later.

Another striking example of long remissions is that of Mrs. L. With the exception of attacks of migraine from which she suffered, she was quite well until her marriage, at the age of 28 years. Two weeks after marriage she had the first attack of which she was aware, and the

⁹ Starr: *Familial Forms of Nervous Diseases*, p. 258.

¹⁰ Weber: *Report of 160 Cases of Epilepsy*, Boston Medical and Surgical Journal, May 25, 1895.

convulsion recurred at intervals varying from four to ten weeks, always during sleep. During her first pregnancy she had but one attack, but this was a prolonged one, in which there was a condition of status epilepticus. At 45 years of age, the attacks, which had lasted for sixteen years, ceased, and there was an interval of 12 years, in which she had none. The menopause did not occur until she was 51 years of age, or six years after the attacks had ceased. When 57 years of age the attacks began again, and there was no assignable cause for their return, except that she had been assiduously nursing a sick mother for a year. When seen by me, in November, 1894, the attacks had become rather more frequent. Under the use of moderate doses of the bromides with belladonna, there was an interval of fifteen months in which she had no attack. The attacks then returned, and occurred about once a month.

Another patient had no attack for an interval of 21 years. The patient was a male, who had his first seizure when he was seven years of age. The attacks were frequent, but only slight. He continued to have the fits, in spite of various forms of medication, including the bromides in large quantities, until he was 17 years old. At that time he was ordered a prescription containing ammonium bromide, tincture of belladonna, and tincture of aconite root. After taking this prescription for a few days, he became violently excited and maniacal. The pupils were dilated, and it was considered that he was suffering from belladonna poisoning. Soon after the medicine was stopped he recovered his normal condition, and from that time, until his 38th year, there was no convulsion of any kind. The attacks then began again, generally in the form of petit mal, but at intervals of a few weeks there was a general convulsion. Another case of interest was that of T. J. K., male. He had no convulsion in infancy, and had sustained no injury to the head. When he was 10 years of age he had an epileptic convulsion one

night. For a year he had no other, but the attacks then began to occur at intervals of one a day to one in two or three months. When he was 26 or 27 years of age, the attacks ceased, and the arrest could not be traced to any medication or change in his mode of life. When he was 41 years old, i. e., after an interval of 21 years, the attacks returned, and when seen four months after the recurrence he was having about two attacks a month.

To these cases may be added the following, in which there was an arrest of the attacks and no return up to the time when the patient was last seen: Edgar S. was 14 years old when he had the first attack. The seizures continued until he was 18 years of age. He had no recurrence of the attacks for five years and four months when last heard from. T. H. M., male, was 20 years of age when he had his first attack. The attacks have always been nocturnal. They continued for 11 years, and then ceased, and when seen a short time ago he stated that he had had no attack for six years. A. R., male, was 9 years of age when he had the first attack. The attacks continued for four years, and there had been no recurrence after two and a half years. A brother of the above was first seized with epilepsy at nine years of age. He had attacks for four years, and had had no recurrence for five years, when last heard from. In some of the above cases the arrest of the attacks could be attributed to medication, but in a few the attacks seemed to cease without treatment.

After consideration of the cases above referred to, in which after prolonged intervals, even as long as twenty-nine years, there has been a recurrence of the disease, we are forced to the conclusion that it is not justifiable to consider any case of epilepsy cured, no matter how great has been the interval of freedom from attacks and appearance of normal health. Notwithstanding this unfavorable conclusion, the study of these cases brings out a fact which is satisfactory, for it shows that remissions of many years' duration may occur, in which the patient

is in normal health, and is able to pursue his life, as if he had never suffered from epilepsy.

DISCUSSION.

Dr. Edward D. Fisher would take the view that most observers do regarding epilepsy—that it probably belongs to the class of incurable diseases; but, while saying this, he did not mean that it was not susceptible of considerable improvement by treatment. The fact of the occurrence of these remissions between epileptic seizures would seem to prove that. He had frequently met with remissions of two, three and five years. Again, he had seen epilepsy occurring in childhood, at the usual period of the tenth or fifteenth year, and then disappearing until some later period of life—e. g., the climacteric, or after some great strain. The value of the paper just presented lay in the clinical demonstration of this peculiarity of epilepsy. This also goes to show how little is really known of the true pathology of the disease.

Dr. Hugh T. Patrick, of Chicago, had seen a long remission in a lady, now about seventy-two years old, who suffered from epilepsy, apparently the result of arterial sclerosis. She had genuine epilepsy as a girl, as shown by her history. This lasted until the time of her marriage; then she had *petit mal* until the time of her menopause, at forty-five years, when the attacks ceased entirely, until the age of seventy, so far as could be ascertained by careful questioning of herself and children. When seventy years old, she began again to have attacks of rather typical epilepsy.

Dr. Patrick said he had now under his care a young woman who had been advised to get married as a cure for her epilepsy. She followed this advice at the age of twenty, and, strangely enough, did not have an attack for two years and a half. She had two pregnancies. This was the only case that he had known of in which there had been any remedial effect from marriage.

Dr. W. L. Worcester thought that, perhaps, in discussing the curability of epilepsy, it would be well to determine just what is meant by the term "epilepsy." It was pretty well known that, on enquiring into the history of epileptics in whom the disease was said to have begun at the age of puberty, it would be found that the patient suffered from convulsions in infancy. If these infantile convulsions were a part of the disease, it would be an example of long remissions, but instances frequently occur in which precisely similar convulsions are observed in infancy for a number of months, and

yet these convulsions cease, and nothing more of the kind occurs in after-life. The question was: Are these to be considered cases of recovery from epilepsy, or are they attacks of an entirely different kind?

Dr. Joseph Collins said he wished to say another word about a case of epilepsy whose history he had presented to the association three years ago. The patient had been operated upon, after he had had two distinct epileptic seizures, Jacksonian in character, the initial manifestation being in the right index finger. He was operated upon 8 months after the first attack, and an area of meningo-encephalitis was extirpated. This was more than 3 years ago, and as yet he had had no relapse.

Dr. Collins had looked over his notes on epilepsy since receiving the programme of this meeting, and had found that out of about 300 cases of epilepsy which had been under his observation, 5 had been practically cured. Although he had seen a greater number of cases, many of them were of little service to him, as they had been seen in his wards of the City Hospital, and had been distributed since then to other institutions. Of 300 available cases, in 5 the disease had ceased for a period of from three to eight years. One was a young girl, who had formerly been under the treatment of Brown Séquard, who seemed to have a magic power in the treatment of epilepsy. From the thirteenth to the twenty-seventh year she had had no return of the attacks. Another case was a young man, whose attacks of epilepsy were in the form of violent headaches during a period of three to five years, after which the typical convulsive form developed. He had lived four years without any further attacks. The treatment in the first case had been stopped for about six years; in the second case it had been stopped for about two years, without any return of the symptoms. The third case was that of a young woman who had had a peculiar form of epilepsy, beginning with the dreamy state described by Crichton Browne, Gowers and others, and followed by what might be called hallucinatory attacks—attacks in which she saw places to which she had never been. This was followed in a few years by typical epilepsy. She had had no attack for three years, but was still under treatment. Another case was that of a young girl, 18 years old, who had now been free from attacks for nearly three years. She was still under treatment.

The treatment in all these cases was what might be termed the orthodox bromide plan. Dr. Collins thought that his statistics were corroborative of the statements which had been made by others concerning the curability of epilepsy. In his opinion, the attacks would cease after treatment in about one

case in twenty, provided the treatment was sufficiently careful and comprehensive.

Dr. Graeme M. Hammond said that in this disease it seemed hardly fair to consider attacks which were ten to twenty-five years apart as consecutive attacks of the same illness. The fact that patients have epileptic seizures does not, like scarlet fever, make the persons less liable to have attacks in the future. There must have been some cause for the epilepsy in each case, but the disease having been recovered from under treatment, there was no reason why something might not have occurred to that individual and reproduced the epilepsy. He would consider such cases as examples of cures with epilepsy developed anew. He had always believed that when a case had gone for three years without any treatment and without any attacks, the case was cured. It was almost impossible to state any accurate time after which these cases should be considered cured, but he would consider a patient certainly cured who had gone ten or eleven years without a seizure. Simply because at the end of that time there was another convulsion, he would not think it fair to say that this was a remission of ten or eleven years' duration; it was rather an example of recovery from the disease and of a renewal of the malady from another cause.

Dr. Sinkler, in closing, said that the point just raised by Dr. Hammond was a very interesting one, and one which he had considered without arriving at any definite conclusion. The conditions in epilepsy were much like those in tic douloureux or in neuralgic affections, where an unstable condition of the ganglia or of the cortex brought about a recurrence of the attacks. In many of these cases there was no assignable cause for the disease.

In his paper he had excluded all cases in which the attacks were Jacksonian, and also cases of infantile eclampsia. He had seen a few cases in which traumatic epilepsy, or epilepsy due to brain tumor, had been relieved by operation, the attacks having been absent for three to five years. Keen, in one of his papers, had stated, that if a patient were free from attacks for five years after operation, he should be regarded as cured.

192. THOMSEN'S DISEASE. A Family History. J. C. Clemesha
Buffalo Med. Jour. 53, 1897, p. 16).

An interesting family history is here presented of an affection, generally attacking the younger members of the family. It shows itself in complete facial paralysis, affections of the limbs and the trunk, and is characterized by loss or diminution of reflexes and of mechanical and electrical excitability of the muscles.

The family chart shows that the disease was most marked in the grandmother. In the second generation the disease was less severe, and in the third still less and at greater time intervals. JELLIFFE.

REPORT OF A CASE OF PURULENT INTERNAL PACHYMEMINGITIS, COMPLICATING MIDDLE-EAR DISEASE.¹

By WILLIAM M. LESZYNSKY, M. D.,

Consulting Neurologist to the Manhattan Eye and Ear Hospital, etc.

E. C., male, 23 years of age, was admitted to the Manhattan Eye and Ear Hospital, February 3d, 1898. For many years he had been the subject of chronic suppurative otitis, affecting both ears. The granulations in the right auditory canal were curetted, and the tympanic cavity thoroughly cleared the day before his admission.

This was soon followed by an evening temperature of 104.20 F. He complained of pain in the right ear and mastoid region, and there was tenderness over the mastoid and along the course of the sterno-mastoid muscle of the same side. For four days these symptoms continued unabated, with the addition of rigors and daily exacerbations of temperature, ranging from 103 to 105.6 degrees, and a corresponding pulse rate from 104 to 120. (See chart.) There was no evidence of inflammation of any of the internal viscera. Repeated examination of the blood showed the absence of plasmodium. Nothing abnormal was found in the urine. The ocular fundi were normal, and the neurological investigation proved negative.

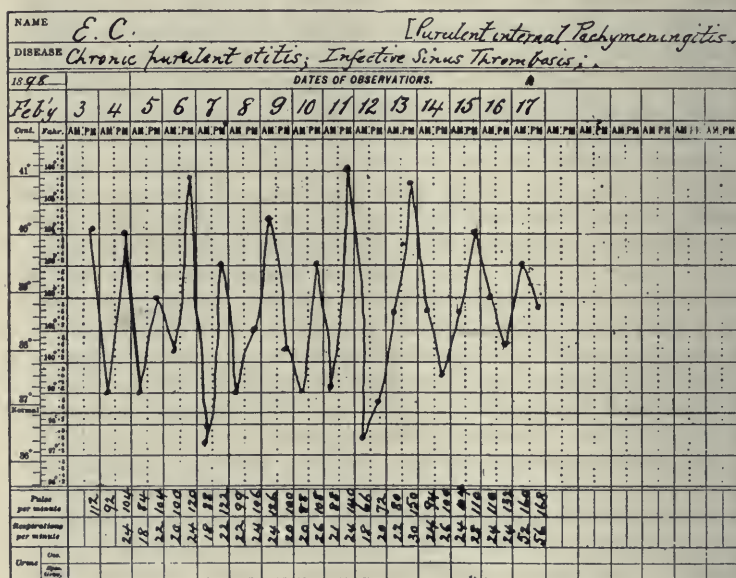
The right mastoid was then opened, and the sinuses explored with negative result. Although the other ear was the seat of chronic suppurative otitis, neither pain nor tenderness was complained of. This was followed by increasing hebetude, rigors at intervals, and temperature at times reaching 106 degrees, terminating in profuse sweating. Vomiting occurred occasionally. On the 15th, inst. (twelve days after admission), it was noticed that he was unable to speak, but could understand what was said to him. I was then asked to examine the patient again.

I found him aphasic, being unable to utter a word, while he understood both spoken and gesture language. It was impracticable to make any further tests in this direction. There was right facial paresis, affecting only the lower branches. The pupils were unequal, the right pupil being dilated to 6

¹Read at the twenty-fourth annual meeting of the American Neurological Association, May, 1898.

mm. Reaction to light was preserved in both. The ophthalmoscope revealed bilateral papillitis with "choked disk" in the left eye. Slight tenderness was present on percussion over the left parietal region, but the patient signified by a negative nod that he had no headache. There was no evidence of mastoid or sinus involvement on the left side.

The right upper extremity was paralyzed, the flexors showing a moderate amount of rigidity upon passive movement. In the right lower extremity, the posterior muscles and the tibial group were parietic. The knee-jerks were exaggerated and ankle clonus was present, both being more marked on



the right side. The cremasteric and plantar reflexes were present, and equal on both sides. The abdominal reflex was absent on the right side, but well marked on the left.

There was no gross disturbance of sensibility. The patient's mental state would not permit of the finer tests.

The continual rigors, high temperature and sweating, in the absence of other indications, pointed to a septic process, most likely the result of infective sinus thrombosis. The provisional diagnosis was made of left temporo-sphenoidal abscess, involving the motor tract, and exploration advised. The operation was performed by Dr. Pomeroy, but no pus

was found. The patient was almost moribund, and required abundant stimulation before being removed from the table.

Within a few hours active delirium developed, and he had to be restrained. Fourteen epileptic convulsions occurred during the night, consisting in conjugate deviation of the eyes to the right, followed by tonic and clonic spasm, affecting only the right side of the face and the right arm. Each attack lasted a minute and a half. The coma increased and death supervened.

Autopsy, 17 hours later. Examination was limited to the head. The external surface of the dura was normal in appearance. Upon opening the dura, the entire convexity of the brain on the left side was found covered with thick foetid pus, but at no point was there any adhesion between the dura and pia. The dura over the left fronto-parietal region was very much thickened and opaque, and contained several hemorrhagic extravasations. The internal surface was the seat of profuse purulent exudation, which evidently originated in its structure.

There was only a very slight degree of leptomeningitis, which was circumscribed over the convexity and involved the left third frontal gyrus, and extended upward about two-thirds of the length of the central convolutions. These cortical areas had undergone well marked softening. The external surface of the left temporo-sphenoidal lobe was sunken inward, and there were masses of clotted blood in the middle cranial fossa, the result of previous surgical exploration.

There was no sign of abscess in either temporo-sphenoidal lobe. The brain tissue and pia were intensely congested throughout. The ventricles were empty, and there was no cerebral oedema. On the contrary, the brain tissue seemed comparatively dry.

The basal ganglia were apparently normal, and no indication was found of softening or hemorrhage in the internal capsule. The right hemisphere showed nothing abnormal. The cerebellum, pons, medulla and the vessels at the base were normal. The usual pyramidal decussation was present. No microscopical examination was made. On the left side the roof of the tympanum was entirely destroyed by caries.

This was probably the source of the pus formation. The sinuses in this region were completely disorganized. On the right side the petrous pyramid was somewhat discolored, suggesting commencing necrosis. The lateral sinus was normal.

In reviewing this case, a few remarks bearing upon the question of diagnosis may prove of interest. Those of

us who have had the opportunity of seeing many cases of middle-ear disease associated with cerebral symptoms, recognize the difficulties that often arise in forming even a presumptive diagnosis as to the location of the cerebral lesion, before localizing symptoms are discoverable.

During the first week several examinations were made with negative results, so far as localization was concerned, and at no time was Gerhardt's symptom (occlusion of the internal jugular vein) demonstrable.

As the previous acute symptoms were limited to the right ear and mastoid, the possibility of the complicating cerebral lesion being on that side, and a non-decussation of the motor tract were also discussed. The condition of the left eye, however, and the fact that the man had always been right-handed, led to the immediate abandonment of such a view.

In order to explain the cause of the rapidly developed aphasia and motor paralysis, several conditions were taken into consideration. Arterial thrombosis, hemorrhage and embolism were excluded for obvious reasons, and the question whether we had to deal with either a left temporo-sphenoidal abscess, or purulent lepto-meningitis, was seriously considered. In the absence of severe headache, delirium, convulsive seizures, etc., which so frequently accompany purulent meningitis, complicating sinus thrombosis, the presumptive diagnosis was made of abscess of the left temporal lobe, involving the motor speech centre and the pyramidal tract.

At no time before operation was headache complained of, and there was only very slight tenderness on percussion over the left parietal region. Neither were any localized spasms manifested, which might lead to the assumption of irritation of the cortical cells in the Rolandic area. It was agreed that all of the general symptoms indicated systemic infection from septic sinus thrombosis, but it is to be regretted that the opposite (left) mastoid and the adjacent sinuses were not explored early in the course of

the disease, as the chronic suppurative otitis was bilateral.

This patient was seen by me in consultation with Dr. O. D. Pomeroy, to whom I am indebted for the privilege of observing and reporting the case.

A CASE OF SEROUS (ALCOHOLIC) MENINGITIS SIMULATING BRAIN TUMOR. BY THEODORE DILLER, M. D. (See p. 441.)

DISCUSSION.

Dr. E. B. Angell, of Rochester, said that about a year ago he had seen a patient who was under treatment by Dr. Koe for middle-ear trouble. Subsequently the patient developed maniacal delirium without focalizing symptoms. The general symptoms were those of septicæmic infection. Choked disk was not present. When the man became delirious, the question of opening the skull and searching for pus was considered, but exploration was deferred from time to time, and the patient ultimately made a full recovery without surgical interference.

Dr. G. L. Walton, of Boston, thought it would have been interesting to have known the order of paralysis in Dr. Leszynsky's case. The rule has been laid down by Macewen that a temporo-sphenoidal abscess working inwards should first affect the leg, then the arm, and finally the face; whereas in case of extension upwards, the inverse order is followed. The case was seen too late to establish this point, but the fact that the arm was decidedly paralyzed, while the face and leg were only slightly so, would tend to show that neither order was followed here, and yet the natural diagnosis was abscess in this locality.

Dr. Walton thought it possible that further experience would teach that this order points by exclusion to meningitis, but the exact diagnosis was baffling at the best, especially when we remember that the process may be metastatic, as well as by direct extension. In the only case of temporo-sphenoidal lesion, confirmed by autopsy, coming under his observation since the publication of Macewen's book, the extension was inwards, and the leg was first paralyzed, thus falling under his rule.

Dr. W. L. Worcester, of Danvers, Mass., said that in connection with the case of Dr. Diller, he was in doubt whether the post-mortem findings accounted for all the symptoms. An exudation of serous fluid into the meninges is frequently misinterpreted: it does not necessarily imply an inflammatory

condition, although it may have done so in this case. The cranium is a closed cavity, and if there is an increase in the quantity of fluid within it, there must be a loss of something else. There was no special thickening of the meninges, and the pathological findings did not account for the symptoms of multiple neuritis.

Dr. F. X. Dercum remarked that it was a well known fact that serous meningitis was apt to be mistaken for brain tumor. In serous meningitis we are liable to have, for some unknown reason, a high grade of optic neuritis. Indeed, this is a much more frequent occurrence than in purulent meningitis. Dr. Dercum thought that the meningitis in Dr. Diller's case could hardly be attributed to alcohol. The whole subject of meningitis serosa was still an open one, but it was a well known fact that its most marked symptoms: diffused headache, optic neuritis and nervous symptoms, vague in character, suggest brain tumor in a silent region.

Dr. Leszynsky, in closing, said that the course of the paralysis in his case would not permit us to exclude temporal abscess, as an abscess in that location might also involve the arm and face fibres more than the leg fibres. While Macewen's views and experience might apply to his own cases, the speaker thought they could not be accepted as an absolute law.

Dr. Diller stated that in his case there was considerable serous exudate, but no pus. Meningeal inflammation was present in spots. The patient gave a very clear history of prolonged indulgence in alcoholic stimulants, and alcoholic neuritis was undoubtedly present. Several writers, among them Quinke, Prince, and Oppenheim, have referred to the similarity of the symptoms of serous meningitis and brain tumor. As a rule, these cases have been mistaken for brain tumors.

193. EFFECT OF STUDY FOR EXAMINATIONS ON THE NERVOUS AND MENTAL CONDITIONS OF FEMALE STUDENTS. Frances M. Drury and Clara F. Folsom (Psychological Rev., 5, 1898, p. 55).

Twenty-five subjects were tested in the following order: (1) For steadiness, (2) for fatigue, (3) for steadiness after fatigue, (4) for memory, and (5) for discriminative ability. The experiments were first made under normal conditions, and again during the mid-year period. The conclusion reached was that "the nervous condition of the subjects was in a slight degree less steady during mid-year examinations than it was normally, but that the mental condition was much improved, thought being more sure and active. Their results show: (1) *In steadiness* 9 were improved, 11 less steady, and 5 unchanged. (2) *In fatigue* 12 improved (greater capacity for mental arithmetic), 8 had less capacity, and in 5 no change. (3) *In memory* 18 improved and 7 were worse. (4) *In discriminative ability* 10 overestimated lines more than normally, 4 underestimated lines more than normally, and 11 kept their normal.

CHRISTISON.

Society Reports.

PHILADELPHIA NEUROLOGICAL SOCIETY.

April 25th, 1898.

The President, Dr. Francis X. Dercum, in the chair.

Dr. F. X. Dercum presented a case of

TABES WITH INTERCURRENT HEMIPLEGIA, WITH RETURN OF THE KNEE-JERK UPON THE PARALYZED SIDE.

The family history of the patient, a brakeman, was negative. He had had one attack of gonorrhœa, but denied syphilis. He married in 1873, and his wife has had four children, all of whom are living and well, and has had no miscarriages. In 1883, while working on a railroad, he was run over by two cars; his body was badly bruised, and he sustained a compound fracture of the skull. He was unconscious two days; was operated upon, and apparently fully recovered.

Some years afterward he noticed that he was becoming unsteady and awkward in his movements, and by the latter part of March, 1896, these symptoms had increased to such an extent that he could accomplish his work only by great effort. He does not recall any other symptoms. He was finally obliged to stop work altogether. He remained in this condition until October 29th, 1896. On this date he went to bed about 8 o'clock, apparently as well as usual. During the night, as he expresses it, he "went crazy"; he lost all power in his right arm and leg, talked with difficulty, and was out of his mind and very violent, attempting to injure persons about his bed. It was necessary to hold him. He remained in this condition about three weeks, after which his mind gradually became clear. When he recovered his senses, he talked with difficulty,

and his right arm and leg were paralyzed, the arm being the most affected. He noticed also that his vision was becoming impaired, and three months after the attack of paralysis the right eye was quite blind. The patellar reflexes were absent. He has remained in this state ever since.

At present the patient walks with difficulty, requiring assistance after every few steps. A right-sided hemiplegia exists, the right arm being semi-flexed and the fingers held in a position of secondary contracture, while the right leg is dragged in walking. A study of the movements of the left arm and leg reveals decided ataxia, especially in the leg. On handling the right arm, very little resistance is experienced in extending the forearm or the fingers. In other words, secondary rigidity is but slightly, if at all, present, although the position involuntarily assumed by the arm is that of secondary contracture. The right leg also is flaccid. On testing the knee-jerks the left is absent, but the right quite marked. No ankle clonus, however, can be elicited, nor can any tendon reaction be discovered in the right arm.

The sensation in the legs is somewhat retarded. An examination of the eyes reveals the presence of Argyll Robertson pupils. The pupils are contracted to two mm. in diameter. The optic nerve of the right eye is completely atrophic, and the left also is atrophied, especially upon the temporal side. Vision in the right eye is entirely absent.

A study of this case permits of no other conclusion than that this is an instance of tabes, in which a hemiplegia occurred, in all probability due to hemorrhage into the internal capsule, and in which the knee-jerk returned upon the paralyzed side. Dr. Hughlings Jackson reported a similar case some years ago. The return of the knee-jerk is explained by Dr. Dercum as follows:

The tonus of the muscles depends not only upon the impulses streaming into the cord from the motor area of the brain, but also upon the impulses streaming into the cord from all the other areas of the cortex, from the basal ganglia, the pons and medulla oblongata. It would seem to be the function of the neurons of the motor area to direct and control these impulses through the lateral tract. The lateral tract being destroyed, it follows that

the impulses streaming into the cord from other sources cause an elevation of the muscle tonus above the normal upon the paralyzed side. It is in this way that we can best account for the rigidity, contracture and exaggerated tendon reactions in ordinary cases of hemiplegia, and this theory certainly enables us to explain the curious phenomenon of a return of the knee-jerk in a case of tabes.

DISCUSSION.

Dr. Wharton Sinkler asked whether Dr. Dercum could give any explanation of the fact that the knee-jerk had not returned on both sides. In hemiplegia, occurring in previously healthy individuals, there is grossly exaggerated knee-jerk on each side, as the result of degeneration in both pyramidal tracts.

Dr. Dercum replied that in ordinary hemiplegia we do not have a degeneration which is absolutely confined to one side of the cord. There are a certain number of fibres which do not decussate, and there are others which, though they decussate, are connected with the so-called sound side of the cord, so that the pathological elevation of the muscle tonus is shared by both sides. In his case the muscle tonus had been sufficiently raised on the paralyzed side to cause a return of the knee-jerk, but not on the "sound" side.

Dr. A. Ferree Witmer exhibited

A CASE OF AMYOTROPHIC LATERAL SCLEROSIS.

The patient was a male of 45 years, temperate, a lathe worker, and with a good family history. He had had typhoid fever seven years previously. His personal history otherwise was negative. His present illness dates from December, 1896. The onset was slow. The first symptom was quivering of the muscles at the base of the left thumb. The wasting, rapidly involving the entire upper extremities, began in the left hand. During the progress of the disease the left arm became entirely denuded of hair, which, however, at the present time is fully restored. For the past nine years he has been sexually weak, but has had no disturbances of bladder or rectum.

At present marked wasting of both upper extremities, especially of the left, is noted. The flexion and extension on the left side are nil. The paralysis is flaccid (type of Ley-

den). The electrical reactions with the galvanic current are quantitatively lessened, being absent over the most atrophied parts. Deglutition and respiration are normally performed. The gait and station are normal. The knee-jerks are plus. Abortive clonus exists on the right side, but clonus is marked on the left side. The elbow jerks are present on the left side. No disturbance of the special senses or of general sensibility is noted. The trophic disturbance and non-involvement of the lower extremities Dr. Witmer considered particularly noteworthy.

DISCUSSION.

Dr. Spiller thought that the case could hardly be considered one of pachymeningitis cervicalis hypertrophica, chiefly on account of the absence of pain and sensory disturbances. Unless we should regard it as one of the rare instances of syringomyelia, without involvement of the sensory fibres, it could hardly be a case of syringomyelia. The symptoms were not those of spinal tumor or myelitis, but were those which we believe result from lesions of the lateral columns and anterior horns of the cord.

Dr. John K. Mitchell said that it is very common in trophic lesions to see both loss of the hair and growth of the hair. Sometimes the growth will be increased at first, or the hair may at first drop out, and then return with increased growth, or the growth may increase late. As to theories, he had none to offer; but all of these conditions he had seen following nerve lesions. He believed that sometimes a part of the increase is due to the rubbing and the applications resorted to as measures of treatment.

Dr. Witmer stated that he had called particular attention to the change in the growth of the hair, because that is supposed to be due to trophic lesions, and trophic lesions are said not to be present in amyotrophic lateral sclerosis.

Dr. J. W. McConnell presented

A CASE OF OBJECTIVE TINNITUS IN A PATIENT WITH GRAVE HYSTERICAL SYMPTOMS.

The patient was a woman, 20 years old. In 1890 a habit spasm of the facial muscles seemed to follow a severe accidental hemorrhage. The affection was migratory, implicating first one, then another group of muscles. She

always had perfect control of the tongue, but had occasional attacks of paralysis of the right lower extremity. This condition persisted for four years, and was followed by a state of perfect health for eighteen months. In 1895, absent-mindedness and loss of memory evidenced a change in her character, and tonic convulsions, general in character, but without loss of consciousness, were added to the symptomatology. These convulsive seizures were of two kinds, one the mother described as "unconscious spells," in which the patient seemed to fall asleep for two or three minutes without muscular movements. The other variety, "conscious spells," were, according to the description, tonic convulsions without loss of consciousness. Shortly after these attacks began three nails of one foot dropped off, apparently spontaneously; those of the other foot seemed ready to drop, but were not shed.

The "unconscious" spells later took on a different form. Consciousness seemed to be lost. There was no spasm, but the patient assumed attitudes of joy, sorrow or prayer, or performed acts suggested by her conversation or employment immediately previous to the onset of the attack. She never fell, never injured herself, sometimes opened and sometimes closed her eyes, recovered almost immediately, and continued her work or talk.

She now has hyperæsthesia of ovarian, mammary and spinal regions. At times there is apparently no secretion of urine, at least, none is passed through the natural passage for as long as forty-eight hours. Then, again, she has polyuria. She says she "has a clock in her head," and by listening a few inches from the right ear a distinct ticking or clicking noise is heard, averaging ninety-four times a minute. It is not synchronous with the pulse, and is audible with the mouth shut or open.

Under hypnotic suggestion the patient has vastly improved, and many of the symptoms mentioned have disappeared. Dr. McConnell thought that the case was one of grave hysteria.

Dr. John K. Mitchell read a paper on

CASES OF TRIGEMINAL SPASM: RESECTION—PROBABLE
PRESENCE OF SENSORY FIBRES IN THE SEVENTH
NERVE. (See page 392).

DISCUSSION.

Dr. Charles K. Mills thought that the first explanation which Dr. Mitchell suggested was more probably the correct one, although it was true that some records indicated a sensory distribution in the seventh nerve. He said that members of the society would probably recall that he had directed attention, and was probably the first to do this, to the fact that both in hysterical and in organic hemianæsthesia we very commonly have preservation of sensation in certain portions of the face on the affected side of the body, that part being usually a part or all of the region here indicated. He referred to a diagram in his book illustrating this. One of his cases was a patient upon whom an autopsy showed a lesion in the thalamus and a portion of the internal capsule. This would indicate that each side of the neuraxis supplies both sides of the face, near the median line, with nerves of common sensibility. Destruction of one side, therefore, would not necessarily cause anæsthesia, or, at least, persistent anæsthesia, on either side in this region.

Dr. Spiller called attention to the very careful dissections made by Zander, which show that the diagrams representing the distribution of the fifth nerve are incorrect. The fibres of the different branches of the fifth nerve go further than we suppose. He had seen cases of facial paralysis in which pain was marked, and thought that it is a mistake to say that the facial nerve is *purely* motor.

Dr. John K. Mitchell remarked that if the explanation of Dr. Mills is the correct one, viz., that both sides of the face are supplied by each nerve, we should not expect to find anæsthesia at all immediately after operation. A careful study of the sensory changes in a sufficient number of cases of facial palsy would help to settle the question under dispute.

Dr. Wharton Sinkler reported

A CASE OF FUNCTIONAL TREMOR SIMULATING DISSEMINATED SCLEROSIS.

A young man, aged 25 years, suffered from an excessive and exaggerated intention tremor. He had an excellent family history; had always been temperate in his habits, and denied any venereal disease. About eighteen months before coming under observation he noticed a slight jerking in the left leg while walking, and, soon after, tremor on voluntary effort was observed in the

right hand. The symptoms became markedly accentuated, in spite of complete rest in bed for several weeks, and, finally, the patient became unable to walk on account of want of control of the right leg. A coarse tremor in the left arm, whenever any attempt was made to move it, was developed to such an extent that he was unable to make any use of the arm. Internal squint, from paralysis of the right internal rectus, also existed.

The patient said that at one period he was unconscious of his surroundings. He held conversation when necessary, but afterward did not remember what occurred during this time. After the tremor had lasted for eight or nine months in the left arm and leg, the right arm and leg became affected, and as the tremor developed in the right side, the left side recovered. He was seen by the speaker in November, 1897. At that time he was able to walk, but required assistance, as incoördination in the movements of the left leg was marked, and as a result there was a tendency to pitch to one side. The left arm and leg were apparently normal. He used the left hand for shaving, writing, and for general purposes, without any tremor whatever. Any attempt to use the right hand brought on excessive tremor, which was identical with the intention tremor of disseminated sclerosis. While at rest no tremor was present in the arm or leg. The kneejerks were excessive, but no ankle clonus was noted. No facial paralysis existed, although the expression was blank. The speech was slow, drawling and scanning, and the patient was easily moved to laughter or tears. His mental condition did not seem to be up to the average. An examination of the eyes revealed no changes in the muscles or in the fundi.

After being under treatment for three or four weeks his condition improved very materially, and he was able to walk about, but soon after there was exacerbation in all of the symptoms, and a return of tremor to the left side. Then tremor developed in the hands while they were at rest. In January, 1898, after a month of absolute quiet and the use of the Paquelin cautery to the nucha, and the administration of hyoscyamus, the patient began to improve again, and became able to feed himself with the left hand, and to walk about with assistance. At the present time the patient's condition is as follows: There is no tremor in either limb while at rest, but any

attempt at movement with the right arm brings on excessive tremor. Slight tremor is still present in the left hand, but this does not interfere materially with the use of the hand. Dr. Sinkler considered the case one of functional tremor, and thought that it was probably hysterical, although many of the stigmata of hysteria were absent.

DISCUSSION.

Dr. James Hendrie Lloyd said that the diagnosis of functional tremor is an important and interesting point in clinical study. He thought that the important question was the elimination or establishment, as the case might be, of hysteria. Where, as in Dr. Sinkler's case, the tremor was shifting in character, passing from one side to the other, the inference was very strong in favor of the possibility of its hysterical origin. He hardly thought that the absence of sensory stigmata and of contraction of the visual fields was positive proof against hysteria. A few years ago he put on record a very interesting case of hysterical tremor, associated with anorexia and great emaciation. In that case the symptoms were shifting, coming and going, with some affection of speech and constant vomiting. That patient made a perfect recovery. She has since married and had several children. Cases of this form of hysteria have been recorded as cases of disseminated sclerosis cured by hypnotism. A French observer, Luys, has claimed that he cured two cases of disseminated sclerosis by hypnotism, with the aid of revolving mirrors. Dr. Lloyd had no doubt these were cases of hysterical tremor, not disseminated sclerosis.

Westphal once described two cases of pseudo-sclerosis, in which the autopsies were entirely negative. Without these autopsies the cases would probably have continued to be regarded as organic. This "pseudo-sclerosis" of Westphal has been justly relegated by French critics to hysteria.

Dr. John K. Mitchell was astonished to hear any one lay stress on the absence of changes in the visual fields as evidence against hysteria. These changes are rather the exception than the rule in hysteria. Not more than one in eight or ten has distinct concentric narrowing of the fields, and not more than one in twenty any change in the color fields.

He had under observation a case of hysterical tremor which resembled in character that described by Dr. Dercum and Dr. Parker as being brought on by continuous slight muscular effort of one kind. The man even had been thrown to the floor by the violence of the general clonic movement resulting from the tremor, which had its origin in the effort to supinate, or to hold supine, the right hand.

(To be continued)

Periscope.

With the Assistance of the Following Collaborators:

CHAS. LEWIS ALLEN, M.D., Wash., D.C. R. K. MACALESTER, M.D., N.Y.
J. S. CHRISTISON, M.D., Chicago, Ill. J. K. MITCHELL, M.D., Phila., Pa.
A. FREEMAN, M.D., New York. H. PATRICK, M.D., Chicago, Ill.
S. E. JELLIFFE, M.D., New York. JOSEPH SAILER, M.D., Phila., Pa.
WM. C. KRAUSS, M.D., Buffalo, N.Y. HENRY L. SHIVELY, M.D., N. Y.
W. M. LESZYNSKY, M.D., New York. A. STERNE, M.D., Indianapolis.

ANATOMY AND PHYSIOLOGY.

194. PRESENT METHODS OF PREPARATION OF THE NERVOUS SYSTEM
H. J. Berkley (Am. Jour. Insanity, 54, 1898, p. 333).

The present article gives an excellent summary of the technique which may be followed in the study of the nervous system. It is a useful and timely résumé.

195. THE INTRACRANIAL CIRCULATION IN SOME OF ITS ASPECTS
George Elder (British Medical Journal, 2, 1897, p. 1,414).

After a number of careful experiments and a full consideration of the work of others, the author comes to the following conclusions:

1. In the unenclosed skull there are two very evident forms of pulsation of the brain—(a) the arterial, (b) the respiratory; the former produced in the arteries, the latter in the veins.

2. In the closed skull the venous side of the circulation is of the greatest importance, and respiration, acting through the veins, takes an important role in the intracranial circulation.

3. During inspiration in the open skull the aspiration of blood from the cranial veins is accompanied by compression of the vessels and retraction of the brain.

4. In the closed skull a similar withdrawal of blood from the veins occurs, accompanied by decrease in the intracranial pressure.

5. There is no flow of cerebro-spinal fluid from the spinal cavity to the intracranial cavity, either with respiratory movement or with arterial pulsation, as has usually been supposed.

6. In all probability, accompanying the emptying of the large veins during inspiration, there is dilatation of the arteries. (This may be accompanied by an increased rate of flow in the carotid artery as compared with the other arteries in the body above the level of the diaphragm, which are otherwise under somewhat similar conditions.) So, during expiration, the dilatation of the intracranial veins will be accompanied by narrowing of the arteries. The flow of blood through the capillaries will remain constant.

7. With arterial pulsation lateral pressure is exerted on the intracranial veins, leading to an increased flow of blood from the skull.

8. These movements of the veins, with respiratory movements and with arterial pulsation, will occur in the large veins leading into the intracranial venous sinuses, the walls of which are themselves incompressible.

9. Similar movements of alternating compression and dilatation of the arteries and veins probably occur also in the spinal column, the cavity of which is to be looked on as being practically closed, just as the cranial cavity is.

10. In all cases of sudden increase of pressure affecting both cranial and spinal cavities there will practically be no variation in the quantity of blood present in the cavities. There will only be either (a) a variation in the amount of blood in one side of the circulation as compared with the other, arterial at the expense of venous, or vice versa, or (b) an alteration in the rate of flow through the capillaries. In rises of pressure lasting some time there may be alteration of the quantity of blood inside the cavities, resulting from alteration in the rate of secretion or of absorption of cerebro-spinal fluid.

11. The point of importance in alterations of pressure inside the skull is the rate of flow through the capillaries. Steady flow through the capillaries—"adiamorrhysis," as it has been called by Geigel—may go on under very low intracranial pressure or under comparatively high. (Hill.)

12. Increased intracranial pressure from an effusion into the cranium—for example, a hemorrhage—leads first of all to flow of cerebro-spinal fluid from the cranial to the spinal cavity, so giving a respite for a certain length of time to the intracranial circulation. If the effusion goes on there is, first, compression of the larger veins, which leads, after it has taken place to a certain extent, to interference with the flow of blood through the capillaries—adiamorrhysis—and, if the pressure still rises, to actual compression of the capillaries and true anæmia of the brain.

13. When tracings are taken of the pulsations of the brain, with rise of pressure two forms of pulse wave may be seen: (1) Where wave is less ample and still anacrotic, as it is under normal conditions; (2) where wave is higher and tends to become katectotic. The former occurs much more frequently than the latter, and seems to be present in what may be termed "passive" increase of intracranial pressure, that is, where the intracranial pressure is increased from alteration of the circulation in the rest of the body.

14. In some conditions the intracranial circulation seems to vary independently of the circulation elsewhere. This would tend to show that, although the blood vessels of the brain are not directly controlled from the general vasomotor centre, there must be some local mechanism for altering their calibre.

PATRICK.

196. I REFLESSI VASCOLARE NELLE MEMBRA E NEL CERVELLO DELL'UOMO PER VARIE STIMOLI E PER VARIE CONDIZIONI FISIOLOGICHE E SPERIMENTALI (The Vascular Reflexes of the Meninges and Brain of Man due to Various Stimuli and Various Physiological and Experimental Conditions). M. L. Patrizi (Revista Sperimentali di Freniatria, 23, 1897).

In an extended article of over eighty pages, copiously illustrated with diagrams and pulse tracings, the author presents a most elaborate and careful study, historical as well as experimental. The conclusions reached by the author are: (1) The reflexes of the blood vessels in man follow the fundamental principles of localization and

extension observed for other reflexes. (2) The localized vascular reflex is more direct than the radiated vascular reflex. (3) The influence of the cerebrum over the spinal centres is manifest in the matter of the vascular reflexes. (4) The time for the vessel reflex for sensitive stimuli is 3 seconds for the arm and 5 seconds for the leg. (5) The reflex for the cerebral vessels for sensory stimulation has a latency not less than the arm reflex for the same stimulus. (6) During sleep vessel reflex action is retarded, diminishing from the centre toward the periphery, and not appreciable in the lower leg. (7) During sleep the movements of the blood in the brain, following stimuli, are probably active and self-regulating reflexes. (8) In the limbs the vascular reflex for sensorial stimuli and for psychic stimuli requires about 4 seconds longer than reflexes for sensitive stimuli. (9) Each sense stimulated gives its own vascular reaction. (10) Some sensorial stimuli provoke vaso-motor reactions with greater force than others.

JELLIFFE.

PATHOLOGY.

197. AZIONE DELLA TOSSINA DIPTERICA SUL SISTEMA NERVOSA. CONTRIBUTO ALLA PATOGENESI DELLA PARALISI DIPTERICA (Action of the Diphtheria Toxin on the Nervous System). Ezio Luisada e Dante Pacchioni, Torino (Giornale della R. Accademia di Medicina di Torino, 61, 1898, p. 77).

These investigations, under the direction of Prof. G. Neya, sought the vulnerability of the nervous tissues to the diphtheric toxins, and injected the toxic material in (1) the cerebral cortex, in the Rolandic area; (2) in the vertebral cavity, near the medulla, and (3) in the sheath of the sciatic nerve. The animal experimented upon was the dog, 13 being under observation, and the effects of the diphtheric toxins obtained from Dr. Belfonti, of the Serotherapeutic Institute of Milan, were studied from a clinical and anatomo-pathological point of view. The results obtained by these investigations may be summarized as follows: 1. The diphtheric toxins, applied directly to the nervous system, provoke a profound lesion at the point of application, characterized anatomically by an inflammation and a degeneration.

2. These lesions are propagated more or less extensively from the point of application.

3. In the dogs not previously immunised by the anti-diphtheric serum, and which had been injected by a dose sufficiently toxic, the phenomena of local reaction and also those of a general intoxication were noted.

4. In immunised dogs the diphtheric toxins provoked constantly alterations of the central nervous system, intense, localised, but of less extent than those produced in dogs non-immunised.

5. The toxine applied directly to the medulla is propagated rapidly in all directions, preferring the posterior columns, the gray matter and the central canal as routes. In consequence of the bulbar invasion death occurred in the animals more rapidly when the toxins were introduced into the medulla than when applied to any other portion of the cerebro-spinal axis. When the toxins were introduced in the cerebral cortex, characteristic lesions of these regions were manifested. Death occurred later, through propagation of the poison to the medulla.

6. Toxines introduced into the sheath of the sciatic nerve provoked an inflammatory process more or less intense, but more circumscribed than in the central nervous system. From the nerve the

toxines ascended to the medulla, chiefly through the posterior columns, and thus provoked an ascending myelitis.

7. The lesions produced upon the neuroglia by the direct application of the toxines are the same as described by Vassale, Donaggio and others in the various intoxications and infective processes. In the oblongata the prevalent alterations are found in the crossed pyramidal tracts and posterior columns.

8. The alterations produced by the toxines affect the nerve fibres more than any other part of the nervous tissue. These lesions affect principally the myelin, and consist in a physical modification of the same, whereby the connections between the various nerves are lost. There is partially a chemical modification of the myelin also present.

9. The local action of the toxines has much importance in the genesis of various paralyses as seen in the human family, attacking first the sheaths of the nerves, then the nerves, then later the nerve centres in the oblongata. KRAUSS.

198. BEITRAG ZUR PATHOLOGIE DER GANGLIENZELLE (Contribution to the Pathology of the Ganglion Cell). O. Juliusburger and E. Meyer (Monatsschrift für Psychiatrie und Neurologie, 3, 1898, p. 316).

These writers conclude, from their examination of a number of cases, that the changes which occur in the chromophilic elements of the ganglion cells are quantitative, vary, therefore, only in intensity, and do not differ in character in the various diseases. They cannot distinguish between the "reaction at distance" and the primary lesions of the cells. The structural cellular changes are simply the manifestations of altered cell vitality. According to their views, the chromophilic elements are capable of regeneration. SPILLER.

199. ZUR PATHOLOGIE DER HEMIPLEGIEN IM GEFOLGE DES KEUCHHUSTENS (Contribution to the Pathology of Hemiplegia Resulting from Pertussis). Hans Luce (Deutsche Zeitschrift für Nervenheilkunde, 12, 1898, p. 272).

A boy of five years became hemiplegic immediately following a convulsive attack in whooping cough. Death occurred after two days. A careful microscopical examination failed to reveal a sufficient cause for the hemiplegia. No hemorrhage within the nervous system was found. Clonic convulsions, especially marked on the paralyzed side, indicated that the paralysis must be of cortical origin, and similar to that occurring in Jacksonian epilepsy. Considerable importance is laid by the author on the accumulation of CO₂ in the repeated convulsive attacks. Luce believes that the hemiplegia occurring in pertussis is due to meningeal hemorrhage, or has no detectable anatomical lesions, and that hemorrhage within the inner capsule or elsewhere in the motor tracts has not been demonstrated as the cause of such hemiplegia. SPILLER.

200. LÉSIONS HISTOLOGIQUES DE LA CELLULE NERVEUSE DANS LE TÉTANOS ET L'IMMUNITÉ ANTI-TÉTANIQUE (Fine Histological Lesions of the Nervous Cellule in Tetanus and Anti-Tetanic Immunity). MM. Chantemesse et Marinesco (La. Med. Moderne, 9, 1898, p. 79).

The idea that the development of tetanus is due to a combination of the tetanic poison with the nervous cellule is not new. The authors have stated in a new fashion the confirmation of the theory

by stating the alterations produced by the toxin in the nerve cells. They examined the alterations in the large cells of the anterior horn of the cord in guinea pigs after a fatal dose of tetanus toxin was given in such proportion as to make it act slowly. Some of the animals received only the toxin, others a mixture of toxin and anti-toxin, others anti-toxin, twenty-four hours after the administration of toxin.

Their final conclusions are that the toxin produced decided lesions in the spinal cells, which might disappear if the animal lived long enough. The mixture of toxin and anti-toxin produced no appreciable symptoms, and the autopsy showed very slight changes in the nucleus and the nucleolus.

The nerve cell has an affinity for the tetanic toxin and the latter for anti-toxin. The precise nature of the cellular reaction cannot be decided. It may, however, be concluded from these observations that immunity to the tetanic poison shows itself in the form of appreciable anatomical changes in the nerve cells. If immunity against infection is due to the action of phagocytes, immunity to soluble poisons is a function of the resistance of the nerve cells, a phenomenon, that is to say, essentially histogenic in character. MITCHELL.

CLINICAL NEUROLOGY.

201. PARALYSIE DOULOUREUSE DU FACIAL NERVE, AVEC HERPÈS ZOSTER DE L'OREILLE (Painful Paralysis of the Facial Nerve with Herpes Zoster of the Ear). M. L. Jacquet (Bulletins et Mémoires de la Soc. Méd. des Hopitaux de Paris, 15, 1898, p. 405).

Jacquet reports a case of left facial paralysis, which had lasted five days. In addition to the well-known signs, he notes:

1. A swelling of the preauricular region.
2. A red and painful œdema of the left ear, on the concha of which a group of herpetic vesicles was found.
3. A very painful point just below the auditory canal.
4. Pain on pressure over all the facial muscles of the left side.
5. Increase in temperature in the skin of the left side of the face.

All these signs developed in one night after the patient had been exposed to a draught. The writer speaks of this as a painful paralysis of the muscles supplied by the facial nerve, with vasomotor and trophic disturbances, probably resulting from exposure to cold. The points of emergence of the fifth nerve were not painful. Jacquet believes that neuralgia of the facial nerve is more common than is usually believed, and may exist with or without paralysis of this nerve. He reported a case of neuralgia of the seventh nerve without paralysis in the preceding number of this journal. He is inclined to believe that sensory fibres are contained in the seventh nerve.

SPILLER.

202. DIE RÖNTGEN-STRAHLEN IM DIENSTE DER HIRN-CHIRURGIE (The Röntgen Rays in the Surgery of the Brain). S. E. Henschen (Mitteilungen aus den Grenzgebieten der Medizin und Chirurgie, 3, 1898, p. 283).

A man was shot through the left eye, and was unconscious three weeks. When he regained consciousness he could neither speak nor understand what was said to him, and was hemiplegic on the left side. Memory and speech gradually returned. About a year after the injury he suffered from headache in the right occipital region. The position of the bullet was determined by the circumstances at-

tending the shooting, by the clinical symptoms and by a Röntgen photograph. The bullet entered through the inner portion of the left eye, and as the revolver was fired from the left side, the bullet was supposed to have crossed the median line. The bullet had evidently gone upward, or otherwise the man would have died from hemorrhage from the large basal vessels, or from injury to the pons and medulla oblongata. Inasmuch as the man was hemiplegic on the left side, and the sense of smell was completely lost, and the chiasm was not injured, the bullet must have passed above the latter, and into the right hemisphere. As the hemisphere was complete, the injury probably was not cortical, and yet the inner capsule could not have been entirely destroyed, as the left-sided anæsthesia was not very pronounced. The vision in the lower left quadrant of the right eye (the left eye was destroyed) was somewhat diminished, and this was supposed to be due to a lesion of the dorsal bundle of the optic fibres. The bullet was believed to be between the parietal and occipital lobes, sub-cortical, and somewhat higher than the calcarine fissure. The Röntgen photograph showed the supposed location of the bullet to be correct, an operation was performed, and the bullet was found on the border of the angular gyrus, somewhat higher than the calcarine fissure. The patient recovered from the operation.

Henschen believes that the dorsal bundle in the occipital portion of the optic fibres (from the external geniculate body to the calcarine fissure) innervates the dorsal retinal quadrant. This has been shown to be true of this bundle in the optic tract and in the calcarine fissure. In this case vision was diminished in the lower nasal quadrant, and the position of the bullet confirmed his theory.

SPILLER.

203. L'ASYMÉTRIE CRANIO-FACIALE DANS L'HÉMIPLÉGIE SPASMODIQUE INFANTILE (Cranio Facial Asymmetry in Spasmodic Infantile Hemiplegia). Dr. Féré (Jour. des Conn. Méd., 16, 1897, p. 363).

Deformities of the face and skull, in infantile hemiplegic subjects, are considered rare by some authors, while by others of common occurrence, which divergence of opinions Dr. Féré ascribes to the difficulty in making correct measurements. These were taken by him in the following manner: 1. Anterior-posterior diameter of the head, from the external occipital protuberance to the tubera frontalia. 2. The dimensions of the orbit, in transversal and vertical directions. 3. The dimensions of the mandibula, from the lateral protuberance of the chin to the angulus. In normal people these measurements are subject to certain variations, but generally symmetrical. In fifteen cases of infantile hemiplegia observed, the author found: Flattening of the tuber frontale on the affected side in 7, equal or increased antero-posterior diameter in the remaining 8 cases; diminution of the orbit vertically in 10, the other 5 cases remaining unchanged. The mandibula on the hemiplegic side showed shortening in 12 cases (80 per cent.), and when well worked gave rise to lateral deviations of the chin and lower jawbone. This atrophy is often associated with a deformity at the junction of the body and angles of the mandibula—a deformity frequently met with in degenerates, described by Albrecht as lemurian apophysis, and supposed to be of atavistic nature, but in reality due to malformation of the teeth and alveolar process.

The ears of infantile hemiplegics present frequently anomalies in this formation, as, for instance, deformity of the tragus, anti-tragus and lobule, and the presence of Darwin's tubercle. This latter may be multiple, and is due to abnormal development, not having any resemblance or connection with the so-called monkey ears, as main-

tained by atavism enthusiasts. The *conclusions* reached in the foregoing considerations are: Certain disproportions and deformities of the face and extremities, developing at an advanced stage of evolution in consequence of a cerebral lesion, have no relation to atavism whatever, nor are the same anomalies necessarily pathognomonic signs of degeneracy. MACALESTER.

204. ZUR DIAGNOSTISCHEN BEDEUTUNG DER LUMBALPUNCTION (The Diagnostic Value of Lumbar Puncture). A. Schiff (Wiener klin. Wochenschrift, 11, 1898, p. 199).

Schiff has studied the literature on lumbar puncture, and added a number of new cases. The procedure has been disappointing therapeutically, but, by affording relief of pressure in such conditions as tumor, hydrocephalus and meningitis, has occasionally been of value in the treatment of pain, convulsions, vomiting, etc. The reports of very favorable results from lumbar puncture in serous meningitis are not numerous. The operation has been of more value from a diagnostic standpoint than from a therapeutic, and has enabled a diagnosis of meningitis, and even its peculiar form, to be made in many cases. It is important to determine whether meningitis is present when symptoms of cerebral abscess or sinus thrombosis develop after middle ear disease, and a cloudy, very albuminous, purulent exudate containing bacteria proves the presence of meningitis, and is a contra-indication to operation. Operation may be performed if the findings are negative, although meningitis is not positively excluded by such findings. Even a large amount of albumin in the cerebrospinal fluid is not a proof of inflammation. Cloudiness of the fluid is a proof of the existence of meningitis. The fluid is clear in all such processes as tumor, abscess and sinus thrombosis, and in many cases of tuberculous meningitis. Negative findings may be obtained in tuberculous meningitis, and occasionally in purulent meningitis. Tubercle bacilli often cannot be found in tuberculous meningitis. Schiff lays great importance on the coagulability of the fluid. Coagulation, when blood is not present in the fluid, indicates a meningitic process even when the fluid is clear. The name of serous meningitis is given to a number of diseases. This condition is found as a complication of otitis media, and simulates cerebral abscess. The coagulability of the fluid in serous meningitis demands more careful study. Schiff believes that lumbar puncture should be employed in every case of meningitis and endocranial complication of otitis media.

SPILLER.

205. "ERGOTISME ET ASPHYXIE LOCALE DES EXTRÉMITÉS" (Ergotism and Local Asphyxia of the Extremities). Mongour (Archives Cliniques de Bourdeaux, 6, 1897, p. 325).

The author describes the case of a woman of 35 years old presenting the following symptoms: When her hands were exposed to cold, they speedily became exsanguinated and white, showing no bluish tint whatsoever. At the same time the tissues covering the backs of the hands, up to the wrists, took on a wooden hardness, not pitting upon pressure, and by their extreme rigidity preventing all movements of flexion. There was no pain, but a sensation of extreme cold in the affected members. The hardness was greater, the lower the temperature: at night softening took place sufficiently to allow flexion of the fingers, and the same effort could be accomplished by day if the patient wore gloves.

The hands were always moist, even when the asphyxia was most marked. There was neither qualitative nor quantitative alteration of

sensibility. The finger nails were diseased, especially upon their under surface, and whitlows had developed upon the left middle finger, the right index and right ring finger. Their evolution was painless, but in such as had cicatrized the scar was painful upon pressure and sometimes spontaneously. Outside of the local lesions, the patient was healthy.

The author concludes that the opinion of Ehlers (of Copenhagen) that symmetrical asphyxia of the extremities is always due to ergotism is not justified, as in the case related ingestion of ergot in any way can be positively excluded.

ALLEN.

206. HEMORRAGIC MENINGÉE (SUS-ARACHNOÏDIENNE PRIMITIVE) SIÈGEANT AU NIVEAU DE LA MOITIÉ DROITE DE LA PROTUBÉRANCE, AYANT PRODUIT PAR COMPRESSION UNE HÉMIPLÉGIE ALTERNÉ DU TYPE MILLARD-GUBLER AVEC PARALYSIE DE L'ABDUCENS DROITE (Meningeal Hemorrhage, Limited to One Side of the Pons and Causing Crossed Paralysis). M. Levet (Lyon Med. 30, 1898, p. 365.

A woman of 74 years was suddenly taken with vomiting and weakness. When seen two hours later the pulse was regular and slow, respiration rapid without stertor, pupils normal, no paralysis but profound coma. The following day there was distinct left hemiplegia, with complete paralysis of the face on the right side. There was no fever, respiration was stertorous and the coma persisted. The next day she regained consciousness for a short time, but rapidly became unconscious again, and remained so until death, five days after the onset. Two days before death paralysis of the right abducens was noticed.

At the autopsy a firm epipial clot was found over the right side of the pons, and there was some slight extravasation of blood over almost the entire brain. The hemorrhage apparently came from the basilar artery. So far as the author has been able to learn, the case is unique.

PATRICK.

PSYCHOLOGY AND PSYCHIATRY.

207. THE PSYCHOLOGY OF READING. J. O. Quartz (Psychological Rev., December, 1897, Supplement).

The author occupies a special number of the Review with the methods and results of his experiments. The results are as follows:

1. Colors are more easily perceived than geometrical forms, isolated words than colors, and words in construction than disconnected words.

2. The visual type of persons are slightly more rapid readers than the auditory type.

3. Rapid readers not only do their work in less time, but do superior work. They retain more of the substance of what is read or heard than do slow readers.

4. Lip movement is a serious hindrance to speed of reading, and consequently to intelligence of reading. The disadvantage extends also to reading aloud.

5. Apart from external conditions the chief factors contributing to rapidity of reading are physiological, intellectual and mental equipment.

CHRISTISON.

208. THE PSYCHO-PHYSIOLOGY OF THE MORAL IMPERATIVE. Jas. H. Lueba (American Journal of Science, 8, 1897, p. 528).

The author contributes an elaborate article on this subject, in

which he lays down the thesis that the "Moral Imperative is the psychic correlate of a reflective cerebro-spinal, ideo-motor process, the efferent end of which is organized into motor tracts coördinated for a specific action." It is not a spontaneous or instinctive act, but a categorical, and thus involves reason and a conscious motive. It is a reflective act, as distinguished from a voluntary act, and, therefore, does not contain "effort" or the provision of the possible motor conclusion, as is the case in voluntary acts. It comes, not unannounced, but unasked for. It is independent of passion, emotion or sentiment. It is the correlate of a purely cerebro-spinal reflective motor process. Emotion and feeling may be an after development.

The "moral" arc is (1) reflective, (2) wholly cerebro-spinal, and (3) it has a clean-cut coördinated motor conclusion prompting to a conclusion. The non-moral arc is the same, but differs in not having an imperative or "oughtness" character. The conclusion of the moral imperative process urges to a specific action affecting some being. The less the moral imperative experience contains an impulse toward the execution of the command, the clearer it is.

"The motor conclusions of a reflective, non-sympathetic imperative ideo-motor experience are always approved of as final." "The moral imperative is the correlate of the latest and highest biological differentiation, since it requires, as a condition of its existence, the independence of the cerebro-spinal from the sympathetic nervous system."

"It appears that the crusade of the ethico-religious consciousness is a war of the cerebro-spinal self against the cerebro-sympathetic self."

CHRISTISON.

209. A STUDY OF THE EXCRETION OF UREA AND URIC ACID IN MELANCHOLIA AND IN A CASE PRESENTING RECURRENT PERIODS OF CONFUSION AND DEPRESSION. C. M. Hibbard (*Am. Jour. Insanity*, 54, 1898, p. 503).

The author presents the following conclusions from an investigation with urea and uric acid excretion in melancholia, based on work done in the McLean Hospital, from 1891 to 1895:

1. The amounts of urine and solids are generally diminished, and they usually increase with the patient's improvement.
2. The specific gravity is normal.
3. The urea and uric acid are, as a rule, diminished.
4. The diminution in nitrogenous excretions is due, in most cases, to a diminished ingestion of proteids, but in some it may possibly result from a lessened absorption of food.
5. The ratio of uric acid to urea shows no constant relation to the mental condition.

JELLIFFE.

210. PARALYTISCHE GEISTESSTÖRUNG IN FOLGE VON ZUCKERKRANKHEIT (DIABETISCHE PSEUDO-PARALYSE). [Diabetes and General Paresis (Diabetic Pseudo-Paralysis)]. R. Landenheimer (*Arch. f. Psychiatrie*, 29, 1896-1897, p. 546).

The author concludes from a study of several cases of his own and a review of the literature:

1. It is not yet proven that general paresis can be caused by diabetes mellitus. The histories, postmortem examinations, etc., of the cases thus far regarded as having been caused by this disease are not exhaustive nor conclusive enough.
2. In some cases of diabetes there is developed a symptom complex, which in many respects resembles some clinical types of general paresis. In the absence of any pathological basis these may be regarded as cases of diabetic general paresis.

In one case of the authors an anti-diabetic treatment resulted in a marked improvement of the patient, thus serving to confirm the relationship in this case. JELLIFFE.

211. MENTAL PHASES OF TUBERCULOSIS. Harriet C. B. Alexander, M.D. (Medicine, 4, 1898).

Alienists not only recognize the *spes phthisica* as an expression of exhaustion, but recognize likewise another symptom, which underlies much of the difficulty in treating seemingly sane victims of pulmonary tuberculosis. This mental symptom, which is so marked that it always arouses suspicion of tuberculosis, as a complication of psychosis at least, is suspicion. The general mental state of the phthisical is essentially that of the primary confusional lunatic plus emotional mobility. There is usually alternating depression, emotional mobility, intensification of the egotism common to invalids, and a suspicious mental state (Spitzka). This suspicious mental state underlies the refusal of and changes in medicinal treatment if the patient be at home, and the refusal of food if he be in an insane hospital. The most decided symptom which appears in the insane in the larval state of the disease is this suspicion. In them, for this reason, physical examination is often difficult, and cough, hectic, etc., are often absent. Frequently a far advanced phthisis comes to a standstill, but demonstrable decrease of the mental symptoms is followed by reappearance of the pulmonary. It is possible to predict tuberculosis from the mental symptoms (Clouston). If these cases have been acute at first, the acute stage is short, and passes rapidly into an irritable, excitable, sullen and suspicious state. There is want of fixity of purpose. The intellect at first is not so much obscured as there is disinclination to exert it. If there be any one single tendency characteristic, it is suspicion. The influence of phthisis on many forms of insanity is to introduce a suspicious element not hitherto present. In some cases the emotional depression produced by phthisis in ordinary types of insanity may proceed so far as melancholia in the true sense of the term. The possible influence of the toxin of the tubercle bacillus is illustrated in the fact that it sometimes causes meningeal tuberculosis to mimic opium poisoning, with resultant coma. FREEMAN.

212. ZUR KATONIE-FRAGE. EINE KLINISCHE STUDIE (A Clinical Study of the Katatonia Question). V. Schüle (Allgemeine Zeitschrift f. Psychiatrie, 54, 1897, p. 515).

The present communication presents an elaborate discussion on the subject of katatonia, first clinically set apart by Kahlbaum, in 1873.

The author does not believe that there is any clinical entity that can with justice be termed a condition of katatonia. The diagnosis is purely then in his opinion a verbal one, including a most irregular collection of motor symptoms. JELLIFFE.

213. KATATONIA (KATATONIE OF KAHLBAUM-KATATONISCHE VERRÜCHTHEIT OF SCHÜLE) F. Peterson and Langdon (Medical Record, 52, 1897, p. 473).

The authors review the literature and report four cases; their conclusions are as follows:

1. Katatonia is not a distinct form of insanity, not a clinical entity.
2. There is no true cyclical character in its manifestations; hence it cannot properly be classed as a form of circular insanity.
3. It is simply a type of melancholia.

4. It is not desirable to retain the name katatonia.
5. The term "katatonic melancholia," or "katatonic syndrome," may be usefully retained as descriptive of melancholia with cataleptic symptoms, verbigeration and rhythmical movements, but should be strictly limited to this symptom complex.
6. The prognosis in melancholia with katatonia is more grave than in any other form.
7. The treatment of the katatonic syndrome is the same as for other types of melancholia.

JELLIFFE.

214. *ALGUNAS CONSIDERACIONES SOBRE EL PRONOSTICA DE LA ALIENACION MENTAL* (Some Considerations on the Prognosis of Mental Alienation). José F. Borda (Buenos Ayres Baletino del Circulo Medico Argentina, January, 1898, p. 13).

Borda studied very carefully the prognosis of the various forms of mental diseases as they occurred in the Hospital de las Mercedes, in Buenos Aires, from 1892 to 1896. Out of 2,350 patients with mental disease, 556 have been cured, 451 improved, 88 have escaped, and 618 have died; a percentage therefore of 23 of cures. In reality this percentage should be greater, because of those escaped some undoubtedly went on to recovery, and of those improved the author believes some were relatively cured. The percentage of cures varies greatly in the different years. For instance, in 1892 it was 17 per cent., while in 1894 it was 33 per cent. In the five years 368 maniacs entered the hospital, and of these 112 were cured, 76 improved, 15 escaped and 104 died. During the same time 300 melancholiacs were received, of whom 55 were cured, 66 improved, 10 escaped, and 59 died. KRAUSS.

THERAPY.

215. *THE DIRECT TRANSPLANTATION OF MUSCLES IN THE TREATMENT OF PARALYTIC DEFORMITIES*. Goldthwait (Boston Med. and Surg. Journal, 137, 1897, p. 489).

The author reports five additional cases of implantation of the lower end of the sartorius into the quadriceps extensor, just above the patella. He says that in acute poliomyelitis, involving the thigh muscles, the sartorius and tensor vaginae femoris are frequently spared. The latter is too small and its range of contractility too limited to be of use as a substitute, but the former is long and powerful. Of the five cases, three showed marked improvement after operation. Not only could the leg be extended with considerable vigor, but the "flinging gait" was largely mitigated, this latter being due, in great part, the author thinks, to the unantagonized action of the sartorius in its normal condition.

PATRICK.

216. *MORPHINE HABIT OF LONG STANDING CURED BY BROMIDE POISONING*. MacLeod (British Medical Journal, 2, 1897, p. 76).

The author reports the case of a lady, aged 25, a victim of the morphine habit for seven years, who by mistake took 18 drachms of sodium bromide in 48 hours. This induced profound stupor, but five days later the bromide was resumed, and continued for three days at the rate of 2 drachms per day. She did not recover from the profound bromism for 10 days, but then found her appetite for morphine entirely gone. Profiting by the experience of this case, "cured by mistake," the author deliberately stupefied his next case of morphinomania with bromide, taking about two weeks to withdraw the morphine and increase the dose of bromide of sodium from 30 grains every six hours to 60 grains every three hours. During the third week the patient was very stupid. The drug was stopped on the 20th

day, after which the patient practically slept for three days, and was unable to stand for a week longer. Five weeks after the cessation of the bromide he had completely recovered from its effects, and had lost all desire for morphine and alcohol.

The following advantages are claimed for this method, based, it must be remembered, on only two cases:

1. It did away with the suffering entailed by stopping the drug.
2. The patient could not bribe the attendants when the drug was withdrawn, he could not deceive his doctor, nor could he escape vigilance—he was powerless.

3. It acted equally well whether the patient wished to be cured or not.

4. No special attendants or establishment were needed; only nurses who took ordinary care.

5. No violence or excitement is likely to result from, nor a taste to arise for, bromide given in this way.

PATRICK.

217. DES ANASTOMOSES TENDINEUSES ENTRE MUSCLES SAINS ET MUSCLES PARALYSÉS POUR LA CORRECTION DES DÉVIATIONS OU DIFFORMITÉS PARALYTIQUES (Tendinous Anastomoses for Paralytic Deformities). Rochet (Lyon Médical, 85, 1897, p. 579).

The author reports 5 examples of this operation, which promises to be of signal value in certain cases of deformity and disability from paralysis more or less limited in distribution. Four of the cases were old infantile spinal paralyses, and one a spastic hemiplegia from infantile cerebral disease. Four operations were done for paralysis and deformity involving the hand and fingers, and one for pes equinovarus. The tendon of a healthy muscle after division was either inserted laterally into the tendon of a paralyzed muscle, or after section of this latter united to the cut end. The results in all of the cases were satisfactory, and in some surprisingly good.

For surgical details the reader is referred to the original, which, on the whole, is an admirable paper.

PATRICK.

218. DE LA VALEUR THÉRAPEUTIQUE DE L'ELECTRICITÉ DANS LE TRAITEMENT DE L'HÉMIPLÉGIE CÉRÉBRALE (The Therapeutic Value of Electricity in Cerebral Hemiplegia). P. Dignat (Bull. Gén. de Thérap., 1897, p. 397).

In quite an elaborate discussion of the subject, the author reaches the following conclusions: 1. In no case of cerebral hemiplegia should electrical treatment be begun for several days subsequent to the attack. 2. At about the end of the third week, electrical interference may be instituted, in which case it should be limited to faradization of the affected muscles for a period of two or three weeks. 3. Then the faradic should be substituted for the constant current, applied along the vertebral regions. The intensity of the galvanic current should be 4 to 5 ma. to begin with, gradually increased, during the course of treatment, to 15 ma., but never higher, and the duration of a séance 10 to 15 minutes, changing the poles once or twice during each application. 4. If the patient shows appreciable evidence of improvement after several days of this treatment, and nothing points to a secondary degeneration, it may be discontinued entirely. However, it is advisable to keep the case under observation, applying static electricity from time to time, in order to keep up the general nutrition, and especially to ward off any functional disorder. 5. In case of permanent secondary contractions, the galvanic current should be used for a long time. 6. No electrical treatment should be prescribed for patients in which the development of focal epilepsy is apprehended.

MACALESTER.

Book Reviews.

ATLAS OF LEGAL MEDICINE. By Dr. E. Von Hofmann, Professor of Legal Medicine and Director of the Medico-Legal Institute at Vienna. Authorized translation from the German. Edited by Frederick Peterson, M. D., Clinical Professor of Mental Diseases in the Woman's Medical College, New York; Chief of Clinic, Nervous Department, College of Physicians and Surgeons, New York; assisted by Aloysius O. J. Kelly, M. D., Instructor in Physical diagnosis, University of Pennsylvania; Adjunct Professor of Pathology, Philadelphia Polyclinic; Visiting Physician to St. Mary's and St. Agnes' Hospitals; Pathologist to the German Hospital, Philadelphia. 56 plates in colors and 193 illustrations. Philadelphia: W. B. Saunders, 1898. Price, \$3.50 net.

This is a valuable addition to the editions in English of the well-known Lehmann's Medical Hand Atlases. The author, the late Prof. von Hofmann, was eminently distinguished in his specialty, and his position of Director of the Medico-Legal Institute of Vienna put at his disposal an almost limitless material. The book reflects the opportunity, as well as the skill, of its creator. The well-chosen subjects, which are taken directly from actual cases, illustrate the appearances and lesions which most frequently come to medico-legal inquiry. Among others, the illustrations bear upon the legal questions which may arise in connection with malformation of the genital organs; with generation; with premature birth and the causes of death in infants; with fractures of the skull and brain injuries; with pistol-shot wounds; with death by hanging, drowning, poisoning, etc. Both the figures and colored plates are accurately and artistically rendered. The explanatory text is ample, and has been translated into excellent English. Altogether, the atlas merits a cordial reception by all who are interested in medico-legal questions.

PEARCE BAILEY.

A MANUAL OF LEGAL MEDICINE. For the Use of Practitioners and Students of Medicine and Law. By Justin Herold, A.M., M.D., formerly Coroner's Physician of New York City and County, etc. J. B. Lippincott Company, Philadelphia, 1898.

One of the largest gaps in the collection of facts with which the average physician's mind is stored pertains to the subject matter of this volume. It is a deplorable fact that so few medical men have even the faintest ideas of the important questions of medical jurisprudence. The present volume is an excellent guide to make good some of the deficiencies. The book is divided in two parts. Part one deals with Toxicology, and part two is devoted to Forensic Medicine. The principal poisons, their effects and lesions, methods of treatment and methods of detecting the same in the dead body are carefully considered. One of the best chapters is "On the Evidences of Death." The medico-legal aspects of some of the recent poisoning cases are well presented.

In Part two on Forensic medicine most every subject of importance to this branch of science is considered, in chapter XX. some excellent ideas relative to the making of medico-legal autopsies being presented. In chapter XXIV. a very good résumé of our knowledge of the blood in its medico-legal aspects is given, and excellent tables of the measurements of various bloods of various animals are prepared. The subject of wounds is exhaustively treated, and the discussion of death from suffocation, electricity, drowning, hanging, etc., carefully considered.

The work closes with an appendix in which a number of illustrative

cases is given. Taking it all in all, the work is a very excellent one, well conceived and executed. ELY.

NORMALE UND PATHOLOGISCHE ANATOMIE DER NERVENZELLEN AUF GRUND DER NEUEREN FORSCHUNGEN (Normal and Pathological Anatomy of the Nerve Cells, based on Recent Investigations). By A. Goldscheider and E. Flatau. Berlin, W. 35. Verlag von Fischer's Medicin. Buchhandlung, H. Kornfeld, 1898.

The authors state in their preface that their work is a critical digest. Our knowledge of the normal and pathological histology of the nerve cells is as yet very limited, and this knowledge has been obtained by widely separated investigators. The value of a digest, therefore, by men so well known as Goldscheider and Flatau, which presents these scattered results within the covers of one small volume, must be apparent to all. On account of the insufficiency of the findings, the abstracts of many papers are given without comment. A short chapter is devoted to the modern technique, and this is followed by five chapters containing most of what is at present known of the normal and pathological conditions of the nerve cells. Little is said regarding the so-called pigments of the cells and the finer structure of the nucleus. Many questions remain unanswered. We cannot decide from this digest, for example, whether the chromophilic bodies are artifacts or not, or whether fibrils exist within the nerve cells. The changes which occur in the nerve cells after division of the peripheral fibres are quite fully described, and many pages are devoted to the action of poisons on the cells. We are not quite sure, however, that all neuropathologists are prepared to ascribe to the Nissl stain the importance accorded by Goldscheider and Flatau. SPILLER.

BOOKS RECEIVED.

"The Insanity Law of the State of New York Revised to April, 1898," by John F. Montignani. Albany, 1898.

"Aphasia and Other Speech Defects," by C. A. Bastian.

"Die Bedeutung der Reize für Pathologie und Therapie," by Prof. A. Goldscheider. J. A. Barth, Leipzig.

"Atlas of Legal Medicine," by E. von Hoffman; edited by Fred. Peterson and O. A. J. Kelly.

"Atlas and Abstract of Diseases of the Larynx," by Dr. L. Grunwald. W. B. Saunders, 1898.

"Insanity," by Clevenger.

"Aix-la-Chapelle as a Health Resort."

"Die Sensibilitätsstörungen der Haut bei Visceralerkrankungen," by Henry Head, M. A., M. D., London.

"Experimental Research upon Cerebro-cortical Afferent and Efferent Tracts," by Ferrier and Turner.

"Zeitschrift für Geburtshilfe und Gynäkologie," by Henry Head, M. A., M. D., London.

"Ueber das Pathologische bei Goethe," von P. F. Möbius. J. A. Barth, Leipzig, 1898.

"St. Bartholomew's Hospital Reports," vol. xxxiii., 1897.

"American System of Practical Medicine," vol. iv., Loomis-Thompson.

"Modern Surgery," Da Costa.

"Atlas of Syphilis and the Venereal Diseases," by Prof. Franz Mracek. W. B. Saunders, 1898.

"Atlas and Epitome of Operative Surgery," by Dr. Otto Zuckerkandl. W. B. Saunders, 1898.

"Mental Affections of Children," by W. W. Ireland.

"Office Treatment of Hemorrhoids, Fistula, etc.," by Chas. B. Kelsey.

THE
Journal
OF
Nervous and Mental Disease

Original Articles.

FAMILY PERIODIC PARALYSIS.*

With a Report of Cases hitherto published.

By EDWARD WYLLYS TAYLOR, M.D.,

Instructor in Neuropathology, Harvard University; Assistant in Neurology, Massachusetts General Hospital; Visiting Neurologist, Long Island Hospital, Boston Harbor.

The condition which forms the subject of this communication is one of great rarity. In America it has, with two possible exceptions, remained absolutely unnoticed in the form which I shall later describe. In Europe, on the other hand, and notably in Germany, a few cases of an identical sort have been reported in detail.

The term "Family Periodic Paralysis" is evidently a clinical one, and serves merely the purpose of description. With certain modifications, this term has been generally used by the German writers, and, in the absence of definite pathological knowledge on the subject, serves its end sufficiently well.

The affection is characterized, in its typical form, by extensive, flaccid motor paralysis, associated with loss of

*Read at the twenty-fourth annual meeting of the American Neurological Association, May, 1898.

reflex and electrical excitability, without sensory or psychic disturbance of any sort, and with intervals of perfect health. The disturbance is periodic, and the predisposition to its development is clearly hereditary. It is at once evident that such an affection has no counterpart among the known diseases of the nervous system, and should, therefore, command our warmest interest. Such interest has by no means been lacking in those who have had the fortune to observe the condition, as a study of the literature amply demonstrates.

The earliest references to a disturbance analogous to that which we are considering come from Cavaré¹, 1853. and Romberg,² 1857. Both of these are referred to by Westphal (5) in a later article, and to his paper we owe the references, which were not obtainable at first hand. Cavaré's case occurred in a woman of 24, who had several attacks of generalized paralysis lasting from five to eight hours; the attacks were of the quotidian type, and were relieved by quinine, but no definite statement is made as to their malarial nature. Romberg describes similar attacks, in which the lower extremities were affected, and which he attributed in his case to intermittent fever. These attacks were cured by quinine.

Nearly twenty years later Hartwig (1), in his inaugural dissertation, again drew attention to the subject through the publication of a carefully observed case of what he called intermittent spinal paralysis. The patient was 23 years old. He had had malaria five years before the present attack, which came on with weakness of the legs, the arms and neck muscles being later involved, somewhat after the manner of a typical so-called Landry's paralysis, only much more rapidly. Speech, swallowing and breathing were hindered. The facial nerve was free;

¹ Cavaré: *Gaz. des Hôpit.*, 1853, No. 89 (aus der *Gazett méd. de Toulouse*), quoted by Erb. *Handbuch d. Krankheiten des Nervensystems*, I., p. 822, als Fall von Macario.

² Romberg: *Lehrbuch der Nervenkrankheiten*, 3. Aufl., 1857, p. 752.

sphincters uninvolved; sensibility and mental state unimpaired. Electrical reactions were almost entirely lost during the attack. Recovery took place in 24 hours. Similar attacks followed, and this patient was also much helped, but not cured, by quinine. He was observed for six months, and the supposition of a malarial cause was entertained. This case, further details of which are given in the summary later, is, in its general features, a type of the condition we are about to study.

Samuelson (2), in 1876, reported a case of intermittent paraplegia, with no disturbance of sensation, in a person of thirty-eight, in which a suspicion of hysteria was aroused, though probably not justified by the facts.

In 1882, Schachnowitsch (3) described a like condition in a patient whose father was similarly affected, and who died from increasing attacks in his fifty-fourth year. The hereditary character of the affection first appears in this observation. In the light of others' experience, death from this cause must be looked upon as unique; this would seem to throw a certain doubt upon the accuracy of the diagnosis, in spite of the fact that in Goldflam's opinion the condition described is accurately classified.

In the same year a paper was written by V. P. Gibney (4), on an unusual form of paralysis, which he regarded as distinctly of malarial origin. He speaks of two cases, both in children, in which, after a malarial attack, paralysis of the extremities came on, associated with muscular atrophy, with certain sensory and constitutional disturbances, followed by slow recovery. The late Dr. Seguin diagnosticated one at least of the cases as myelitis of the anterior horns. Gibney regarded them as similar to the conditions reported by Cavaré, Romberg and Hartwig, to which allusion has already been made. In any event, they are so clearly unlike the typical condition, to which we wish to draw attention, that in our opinion they almost certainly do not belong in the same category, whether malarial or not. They are alluded to in this connection

because Westphal, in a subsequent communication, following his earlier paper, is inclined to regard Gibney's first case as belonging to the group of the family periodic paralyses. Westphal further thinks that neither of Gibney's cases was due to malaria.

The first really important consideration of the disease, if we may so call it, we owe to Westphal (5), who, in 1885, described in detail a typical case under the heading "Ueber einen merkwürdigen Fall von periodischer Lähmung aller vier Extremitäten mit gleichzeitigem Erlöschen der electrischen Erregbarkeit, während der Lähmung." In the study of this case Westphal was assisted by Oppenheim, to whom we owe much careful work on the electrical changes. The history, in outline, is as follows: The patient was a boy of twelve, with no hereditary predisposition, so far as learned. The attacks came on by weakness of the legs; three hours later the boy could not stand, and the attacks were followed in a short time by a complete paralysis of arms and legs, of a flaccid character. Knee reflexes at times were not obtainable. Cranial nerves were free. Sensation was unaffected; mind clear. Electrical examination showed distinct quantitative diminution to both galvanism and faradism, and complete loss in certain nerve distributions. Gradual improvement occurred the following day, which soon became complete, with a slow return of electrical excitability. He was perfectly normal in all respects between the attacks. Repeated attacks of this character occurred, which were carefully studied until the patient passed from observation. The condition above briefly described excited Westphal's profoundest interest. His astonishment was chiefly aroused by the extraordinary electrical and reflex changes, which at once placed the disturbance without the pale of the so-called functional diseases, notably hysteria. Westphal's since much-quoted words on the subject are: "Dieses relativ schnelle Erlöschen und Wiederkehren der electrischen Reizbarkeit in Nerven und Muskeln steht ganz einzig in seiner Art da; wir ken-

nen weder eine Krankheit des Rückenmarks noch der spinalen Nerven, in welcher jemals etwas Aehnliches beobachtet wäre; ebenso lässt uns die Physiologie in Betreff einer Erklärung vollständig im Stich * * * Wir stehen somit dem geschilderten Krankheitsfalle als einem Räthsel gegenüber, und sind nicht einmal im Stande, eine annehmbare Hypothese aufzustellen, weder über die Natur der in grösseren Intervallen (nicht nach dem Wechselieber Typus) auftretenden Lähmungserscheinungen, geschweige denn über die Ursachen des schnellen Erlöschens und der ebenso schnellen Wiederkehr der electrischen Erregbarkeit der Nerven und Muskeln."³

Following Westphal, in the same year, Fischl (6) adds another interesting observation, which, although differing in certain respects from other reported cases, probably belongs to the same general group. The patient was a girl of eight, also without hereditary history of significance. There was a complete flaccid paralysis of the legs, with loss of knee-jerk, the arms being very slightly, if at all, affected; recovery in twenty-four hours. At a subsequent attack, faradic examination showed a complete loss of reaction in muscles and nerves involved. Condition between the attacks perfectly normal. Certain peculiarities of this case are of special interest. These will be hereafter discussed.

In the following year, 1886, Westphal (7) published a supplementary article, in which he discussed Gibney's paper, which he had before overlooked. Beyond this, the communication adds nothing to our knowledge.

Cousot (8, 9), in 1886, and again in 1887,⁴ gives histories of a number of cases occurring in one family. The mother and four children were affected; beyond this the heredity does not go. The mother often lost her motor power once a week, an affliction which lasted through life.

³ Westphal: Ref. 5, p. 511.

⁴ We quote only from the latter article. Ref. 9, of bibliography.

The children suffered from similar attacks, motor weakness to point of paralysis, arms and legs both affected, loss of electrical reactions; sensation and intelligence undisturbed; normal between attacks.

Griedenberg (10) adds another case to the list, that of a man of twenty-two, who had periodic attacks of an identical sort with those already described.

In 1890 Goldflam (11) first published his investigations on the subject with the title, "Ueber eine eigenthümliche Form von periodischer, familiärer, wahrscheinlich auto-intoxicatorischer Paralyse." With the attention which Westphal had already brought to the matter, this and the later work of Goldflam and Oppenheim afford us what knowledge we have of this unique affection. To Goldflam, however, in particular, belongs the credit of working out in minutest detail the clinical phenomena, and also of instituting various experimental researches in the hope of elucidating the subtle problems of etiology. This first complete description of the disease covers eleven cases in the patient's family, and affords, therefore, an opportunity of noting the remarkable hereditary character of the disturbance. The attacks in the best observed of these cases occurred in a boy of 17, and conform in all essential regards with the cases already briefly described. The following year, 1891, Goldflam (13) further elaborated his paper in a detailed piece of work under the same title. This second article covers all the ground of the previous one, and adds considerably to it. These papers, particularly the second, are monographic in their completeness.

Between the publication of Goldflam's two papers Pulawski (12) reported a perfectly typical case, so like others that the same general description serves for all.

In the same year that Goldflam's later communication appeared Oppenheim (14) had an opportunity to observe again the case described by Westphal in 1885, and to make a much more careful examination than had hitherto

been done. The chief value of this work lies in the investigation of the electrical changes, under varying conditions and in different stages of the attacks, as well as in the free intervals. His conclusions in general are that at the height of the attack nerve-muscle excitability is completely lost in certain groups, but that electrical stimulation does not always give uniform results. Oppenheim also draws attention for the first time to a hardly less remarkable phenomenon, viz., a temporary dilatation of the heart during the attack, and signs indicative of mitral insufficiency, which gave place to perfectly normal conditions between the attacks.

An interesting but cursorily reported case appeared in the *University Medical Magazine* for 1892-'93, observed by Burr (15). The patient was a strong man of thirty, who since his tenth year had had periodic attacks, occurring about four times yearly, and lasting from a day to a week. The long duration of the attacks, and the fact that but one half of the body was affected when seen by Burr render the case noteworthy. No electrical examination was made.

In 1894 two papers were published, by Hirsch (16) and Rich (17), of Ogden City, Utah. The mother of Hirsch's patient, who was a man of twenty-six, had similar attacks; otherwise no hereditary predisposition. Hirsch verified Oppenheim's observation of temporary insufficiency of the mitral valve during the attacks. In other respects, the case is only noteworthy from its similarity to others.

Rich's (17) communication is an exceedingly interesting one, and the subject is well deserving of more study than he has devoted to it in his published article. He describes, without citing individual cases, a peculiarity existing in his own family for five generations, of a temporary motor paralysis due to cold, affecting by preference the facial muscles, without accompanying disturbance of the mind or of sensation. An unusual exposure to cold

or dampness brings on almost immediately a more or less complete disturbance of motion. He speaks of one occasion in which a person was completely paralyzed, with the exception of the tongue, through sleeping in moist underclothing. A paralysis of the tongue could easily be brought about by holding snow in the mouth, taste being unaffected. Complete recovery always takes place, preceded by a feeling of exhaustion. The paralysis is always of the nature of a tonic spasm. No mention of temporary electrical changes or of a detailed physical examination is made in his paper. Apart from the peculiarity mentioned, there was absolutely no neurotic tendency in the family, but this one idiosyncrasy had never skipped a generation, as far back as could be traced.

The analogy to the condition we are considering is so striking, and the exciting cause so definite and yet so different from other reported cases, that this paper of Rich must be regarded as most significant and important, in spite of its brevity.

In 1895, Goldflam (18) carried his researches still further, and attempted by various means to arrive at definite conclusions of an etiologic sort, with what success we shall have occasion to see later. In a footnote to this elaborate paper, he alludes to two other cases, in addition to those, eleven in number, which he had previously described, both occurring in children, and he thinks of the same character. He does not go beyond a very general statement, pending further observation. It may also be said, in passing, that Goldflam regards a case, alluded to in Erb's monograph on Thomsen's disease, by Rothe⁵ as belonging to the family periodic paralyses.

Bernhardt (19), in the following year, writes on two cases in a father and son, which correspond closely, in certain respects, to the clinical picture already frequently

⁵ Rothe: Statistischer Sanitätsbericht über die Königl. Preuss. Armee, u. s. w., für 1879-81. Berlin, 1882, p. 51 (ref. taken from Goldflam's paper, bibliog. 18).

outlined. Bernhardt, however, is chiefly interested in the association of periodic paralysis, with muscular dystrophy and with other forms of well recognized disease.⁶

Goldflam (20) contributed still a fourth paper to the subject in 1897. In it he reports eight additional cases, in a branch of the family he had already described, and also three other cases occurring in a totally different family. His conclusions from this further study are essentially confirmatory of his previous observations, though differing somewhat in detail.

From the foregoing historical sketch, which, we believe, is complete in its references, it is evident that the condition outlined is of curiously infrequent occurrence, and that when it has been observed it has aroused an interest of the profoundest sort. The German cases have been studied with extreme minuteness by Westphal (7), Oppenheim (14), Goldflam (11, 13, 18, 20) and others; in America we find but three references, one of which, that of Gibney (4), is doubtful of classification. Of the other two, Burr's (15) communication narrates a single case, which is, no doubt, of the periodic type, but reported with less completeness than its importance warrants; and, finally, the extraordinary condition described by Rich (17), as due to exposure, presents a most interesting and close analogy to the matter under discussion. We have, therefore, no excuse to offer for the presentation of the following family history, and the somewhat detailed consideration of the affection as it occurred in two of its members:⁷

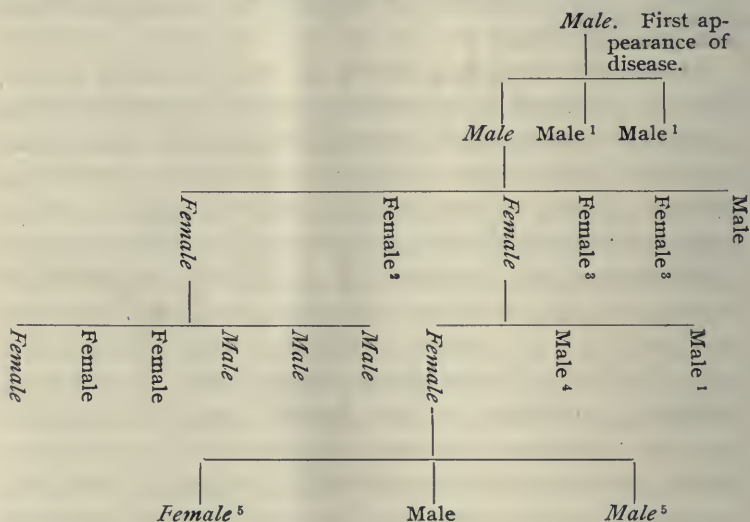
The affection in this family first developed five generations back. There is a tradition that the person afflicted was running violently, when something seemed to give

⁶ See earlier paper. Bernhardt: Muskelsteifigkeit und Muskelhypertrophie (ein selbstständiger Symptomencomplex). *Virchow's Archiv*, 1897, lxxv., p. 516.

⁷ For many of the following facts and observations I am indebted to a member of the family, a student of medicine, whose assistance has been of the greatest help in preparing the clinical side of this report.

way in his back, and he fell to the ground, prostrated. From that time on he was subject to attacks of a periodic character. Of his three sons, only one seems to have had the disease. The other two, with their children, were never afflicted. The son who was affected was a judge, and those who remember him cannot recall that he was ever kept from his work by the attacks, but remember

FAMILY PERIODIC PARALYSIS.



EXPLANATION OF CHART.

- Italics:* Persons affected.
 Roman letters: Persons exempt.
 1. Children exempt.
 2. Unmarried.
 3. Died young.
 4. Epilepsy.
 5. Patients studied.

that he frequently needed assistance in getting in and out of his carriage. In each succeeding generation the disturbance was transmitted to one or more members down to the present, in which two (our patients) out of three are typically affected.

The genealogy of the family is presented in the above diagram, which makes a detailed description superfluous:

The hereditary tendency is transmitted through the female as well as through the male line. Apart from this affection, there is no significant neurotic tendency observable. One person, who was exempt, had epileptic convulsions, and the mother of our patient, and, possibly, the grandmother, died of tuberculosis of the lungs. Neither of these facts is significant, inasmuch as the paralytic condition had already existed for three generations in a perfectly non-neurotic family, so far as ascertainable. It is to be remarked, further, that the family is one of rather unusual mental power.

Detailed facts regarding previous generations are difficult to obtain, although certain members of the second generation are living. The testimony of those members of the family with whom I have been able to talk is, however, definite, viz., that the attacks in all cases, with one exception, when they began at the eighteenth year, have come on at about the age of puberty, and persisted, with varying frequency, during the active years of life, after which, between the fortieth and fiftieth year, they have gradually disappeared. The character of the individual attacks has been in all cases strikingly similar, to such a degree that one description serves for all.

That description we shall give in the following most carefully observed case in the present generation:

CASE I.—J. T. S., 19; schoolboy. As a young boy he was perfectly well, and free from the ordinary diseases of childhood; no malaria nor venereal infection. During the summer of 1894, when he was 14 to 15 years old, he first noticed a certain slight feeling of weakness in the thighs and calves of the legs. His earliest remembrance is that in playing "leap-frog" he was weaker than usual. This condition continued in intermittent fashion during the summer, and in the fall of that year he had the first typical attack. It occurred at night, as all the succeeding ones, practically without exception, have done. The attack was characterized by a general weakness of the body and limbs, without further disturbance of any sort. From that time the attacks followed each other rapidly, on an average of one a week. He would awake in the morning to find himself paralyzed; he thinks he was often awakened

by the paralysis. His helplessness usually continued during the day, and passed away gradually toward night. His own observation was that the attacks were brought on by excitement, exercise or late hours. Between the attacks he was absolutely well, with no trace of muscular weakness.

From that time to this, a period of nearly four years, there has been no change, with the possible exception that the attacks have increased somewhat in frequency and diminished in intensity. This is the general history of the affection, as it appeared to the patient's own observation of himself. Being an active boy, with a taste for athletic exercise, it goes without saying that he has been exceedingly annoyed by his persistent periods of disability. So many of his ancestors, including his mother, have, however, been similarly affected that he has developed a certain philosophical attitude, which helps him over days of enforced idleness.

A typical attack, in detail, is as follows: After a day usually of somewhat violent exercise, as, for example, bicycle riding, he goes to bed feeling perfectly well and strong. There are no prodromal symptoms whatever, beyond, on occasions, a certain feeling of weakness, which usually is not observed, since the attack begins almost invariably at night. The portion of the body first affected, whether legs or arms, is also not definitely known for the same reason, though he thinks a weakness of the legs is antecedent. On awaking in the morning, he finds himself in a pronounced attack, completely paralyzed, with the general exception of the cranial nerves. Voluntary movement is absolutely abolished. Coughing and sneezing are impossible, but respiration, so far as he has observed, remains normal. There is no dyspnoea. The muscular system is perfectly flaccid. He has no desire for food or drink during the attack, and he often goes twenty-four hours or more without urination. There is sometimes apparent temporary urinary retention, which he thinks is due rather to the awkward position in which he happens to be lying than to any actual disturbance with the sphincters. He never defecates during an attack. He has no sensory disturbance whatever, and his mind is perfectly clear throughout. After lying in this helpless condition for hours, he gradually recovers the lost motor power, and toward evening of the same day is able to get up, the power usually returning first in the arms, after which a complete return to the normal takes place. Speech is always unaffected; there is no difficulty in swallowing and never the slightest pain.

From this time until the onset of the next attack the patient regards himself, and his friends regard him, as a normal individual, capable of the same muscular exertion as his companions. He has always noticed that any overexertion tends

to bring on an attack, which must, however, be preceded by a period of rest. In this way he has come to predict, with relative certainty, the occurrence of a "lameness," as he calls it. He usually pays the penalty for a particularly enjoyable day of activity by an enforced rest of many hours the following day, the onset of the paralysis coming some time during the night. He is able to abort a threatened attack temporarily during the day by increased muscular exertion, but it is merely a postponement of the inevitable paralysis, which follows with all the more certainty later. He is not able to attribute the attacks to dietary errors, or to anything whatever beyond the mere fact of exertion.

Physical Examination.—I first saw the patient April 8th, 1897, between attacks. He presented a picture of perfect health, and the examination, as made at that time, showed nothing which could be regarded as abnormal. His muscular system was particularly well developed; the legs somewhat out of proportion to the arms and body. His calves were noticeably large. Measurements made at the time were as follows: Chest under the nipples, $31\frac{1}{2}$ inches unexpanded; $33\frac{1}{2}$ expanded; biceps, midway between shoulder and elbow, right, $11\frac{1}{2}$; left, $10\frac{3}{4}$ inches. Thigh, a little above patella, right, $20\frac{1}{2}$; left, 20 inches; calf, maximum circumference, right, $16\frac{1}{2}$; left, $16\frac{1}{4}$. No pathological significance was to be attached to these measurements.

June 18th, 1897. Saw the patient again at his home, which was some distance from Boston. The previous day he had ridden fourteen miles on a bicycle and otherwise exerted himself, but went to bed feeling well. He awoke at 6 the following morning, completely paralyzed, except for the slightest possible movements of the hands and feet. The face was unaffected. He remained in this helpless condition until about three in the afternoon, when a gradual improvement began, first in the arms. Owing to the distance at which he lived, I was unable to see him until about five in the afternoon, after improvement had begun. He was then in a state of partial paralysis. Movements were made with the greatest difficulty and weakness. The legs were almost completely beyond his control; the arms less so; neck muscles very weak. He could raise himself in bed only with the greatest difficulty. His efforts reminded me of a person in the last stages of a prostrating disease. His mind was perfectly clear, and he made no complaint of pain. It was a hot day, and he was sweating profusely. His face was somewhat flushed, but his temperature was normal. Pulse 88, and of fair quality. The heart^a sounds

^a In view of Oppenheim's work, a more careful examination of the heart is to be desired.

were normal; the pupils were equal, of normal size and good reaction; swallowing was unaffected. He was totally unable to hold his head up, unsupported. All movements of the arms extremely weak; when he was supported in a sitting position he could not raise his arm above the horizontal. The legs were much weaker than the arms. Slight flexion at the knees was possible, and extremely slight flexion at the ankle. Flexion and extension of the toes possible, but very weak. Feet slightly adducted. Muscles all felt normal. Difficulty in coughing and sneezing.

Reflexes: Knee-jerks lost; no plantar; cremaster very slight; abdominal and epigastric, active.

Electrical Examination; the faradic current alone available: Marked quantitative diminution of reaction from the facial nerve. Right arm: median, ulnar and musculo-spiral (?) nerves tested. Slight reaction from very strong currents. Same from Erb's point. Muscles: Deltoid, sterno-mastoid, trapezius, flexors and extensors of forearm, interossei of the hand, excited to contract only by very strong currents. Right leg: Very slight reaction from peroneal nerve; none from the posterior tibial nerve. Muscles: Vastus externus, quadriceps, interossei, slight. Left leg: Even less reaction obtained from the same muscles and nerves. The strength of the current used was sufficient to produce painful tetanic contraction in a normal person.

The result, therefore, of the partial electrical examination, which was alone possible, was enormous quantitative diminution to both indirect and direct stimulation. No qualitative change to faradism was noted. There was no disturbance of any form of sensation, and the patient took an intelligent interest in the examination throughout, answering all questions with perfect clearness.

At six in the evening, twelve hours after waking, he had eaten nothing but a few bananas, and had not had the slightest desire for drink. He had not passed urine for twenty hours, nor had an operation of the bowels for at least forty-eight hours. An hour later, at about 7 o'clock, he was again examined, and it was found that an increase in the weakness had again come on, an unusual occurrence. It was probably to be explained by the effort of muscular exertion, occasioned by the preceding somewhat prolonged examination.

Two days later, June 20th, patient wrote the following letter, which I reproduce in part: "I thought I would write and tell you how I came out of my immovability. When you left I had grown somewhat weaker than when you came, as you knew, but that was only a warning of what was to come. In the night it came back full force. I was unable to sleep very long at a time. It is not exaggerating to say that I was

truly miserable until it left me, about 3 or 4 o'clock Saturday morning. I was so weak and sore that I did not get up until about 11; the rest of the day I took life fairly easy, but was a little tired when I went to bed. This morning (forty-eight hours after first onset) when I woke up, I was a little lame, but after I got up it worked off, and I am feeling quite well now."

The fact of his being able to write in a steady hand shows the complete disappearance of the paralysis from the hand muscles.

Several months later, September 19th, 1897, his aunt, with whom he lives, wrote as follows: "I thought you might be interested to hear of J.'s condition since we were at your office. The following day, Wednesday, he was unable to dress until 3 P. M.; Thursday, not until 5 o'clock; Friday, at 10 o'clock, so went to school at recess. Saturday he was all right; to-day (Sunday), able to get out of bed at 3:30 P. M. He wished very much for me to send for you to-day, etc. Mrs. G.[the sister about to be described] was also in bed the greater part of the day yesterday, only able to sit up about an hour the latter part of the afternoon."

May 11th, 1898, the patient J. was again seen during a free interval. A careful examination resulted negatively. The attacks had diminished somewhat in frequency for the past few months. He is to-day feeling perfectly well.

Physical Examination: Muscular system apparently normal; no impairment of strength anywhere. Mechanical irritability of muscles, normal. Knee-jerks present and normal. Peripheral reflexes obtainable, and sufficiently active.

Sensation undisturbed; pupillary reactions normal; no enlargement of the spleen.

Heart perfectly normal as regards size and sounds. Pulse, 88, good quality.

Electrical Examination: Strong currents well borne. Considerable skin resistance. *Faradism:* Prompt and normal reaction from nerves and muscles of arms examined. Good reaction also from nerves and muscles of legs, with the possible exception of the peroneal group in the right leg. Inasmuch, however, as the indirect stimulation from the peroneal nerves produced reaction of the muscles supplied by it, we would not lay particular stress upon the poor response of the muscles to direct stimulation. *Galvanism:* Normal reactions, with the same exception above noted. Response quick; cathodal contraction much greater than anodal. No polar changes nor slightest indication of a reaction of degeneration. No exhaustion of muscular contraction through prolonged stimulation. In general, somewhat stronger currents were necessary than in case of the control used, but not more than could be

explained by a heightened skin resistance.

Blood: Examination of the blood was kindly made for me by Dr. H. F. Hewes, of the Harvard Medical School. His report of a specimen taken between the attacks is as follows: "Probably no increase in white corpuscles. In a differential count of 200 white corpuscles, basophiles 51%; polymorphonuclear neutrophiles, 47%; polymorphonuclear eosinophiles, 2%; no myelocytes nor other pathological forms. Summary: Lymphocytosis, no evidence of anæmia. The large proportion of basophiles is characteristic of a reduced condition of nutrition. The count would be normal for ten years of age."

In a personal note Dr. Hewes writes: "The examination of the blood of Mr. S. shows practically no evidence of anæmia or of degenerative changes in the corpuscles. The lymphocytosis suggests lack of vigor in the metabolic processes, that is, if the patient is over 10 years of age. It is a condition similar to that found in the blood of rachitis of strumous children," etc.

An examination a few days later, also in a free interval, showed in a differential count of 200 white corpuscles: Basophiles, 57%; polymorphonuclear neutrophiles, 42%; polymorphonuclear eosinophiles, 1%; a few endoglobular poikilocytes and microcytes were seen. No malarial plasmodium. Very slight suggestion of loss of corpuscular substance in red corpuscles, evidently leucopænia plus lymphocytosis.

Of this specimen Dr. Hewes writes: "The relative increase in the younger forms of leucocytes is more marked than in the other specimen. The form is mostly that of the small lymphocyte. There is evidently a very small number of white corpuscles, perhaps four thousand per c.m. of blood. This also is a sign of lowered vigor."

Urine examination between attacks, also by Dr. Hewes; I append his report:

"May 19th, 1898, mixed twenty-four-hour specimen. Quantity of urine in twenty-four hours, 1,170 cc.; color, normal; specific gravity, 1.022; reaction, acid; amount of sediment, slight; urophæen, normal; chlorides, low; indoxyl, increased; sulphates, normal; urea, 1.85%; earthy phosphates, diminished; uric acid, normal; alkaline phosphates, diminished; albumin, absent; sugar, absent; bile pigments, absent; no acetone; total chlorine, 8.5 gms.; total urea in twenty-four hours, 21 gms.; sediment, centrifugal; few small, round epithelial cells; no casts found; a fresh specimen examined one week ago showed nothing abnormal.

"The urine in this case suggests a somewhat low condition of metabolism. The urea excretion is below normal for a man of this age and weight upon ordinary diet. It should

approximate 30 grammes instead of 20. The twenty-four-hour amount of urine is low. The chlorine is low; normal phosphates are low; there is no special evidence of an excessive formation of uric acid in place of urea, nor of failure in carbohydrate metabolism; no sugar or acetone. Proteid metabolism is, as I have said, low. Fat metabolism can, of course, be judged by the stools only."

Owing to the distance at which he lives repeated examination of the patient during the attacks has not been possible. From the foregoing it will appear that the attack described was a severe one of a typical sort, and that the examination was made toward its close, or at least after the most extreme paralysis had begun to pass off. The signs observed were characteristic for that period, the most important being loss of knee-jerk, and great quantitative diminution in electrical excitability.

CASE II.—Mrs. G., sister of previous patient. This patient I have been able to see but once, and then between attacks. She gives the following history:

She was at the time of the examination, September 14th, 1897, 24 years old; married; no children. When she was twelve years old, and much worried about the illness of her mother, on rising one morning she found it difficult to walk. The calf muscles seemed chiefly affected; she could not raise her heels readily. She went downstairs and fell from weakness, or stumbling, she does not know which. She made no mention of the feeling of weakness, and walked a considerable distance, experiencing the same difficulty. This continued during the day, and passed off the following day.

Two or three weeks later she had a second attack, which she attributed to taking medicine by mistake the evening before. In the morning she had the same trouble in walking. Toward night she could not walk upstairs, nor lift her feet. She was carried. The arms and face were unaffected. From this time on she had similar attacks, varying in frequency from one a week to one a month, and growing gradually more severe. A few months later she was completely helpless for the first time, the arms as well as the legs being affected. In the very worst attacks, of which she has had three or four, the face has been somewhat involved. She describes the disturbance as a drawing sensation in the lower part of the face, associated with a certain difficulty in opening the jaws, but not in closing them. She has never been unable to close her eyes. When her face was affected, she also had difficulty in breathing. She could not cough or sneeze. There was no disturbance with the special senses, nor with sensation generally.

Two years ago she took a "headache medicine" of unknown

character in the evening. The following morning, at 9 o'clock, she suddenly lost consciousness while lying on the bed. She found herself about ten minutes later in a corner of the room. She did not bite her tongue, and had never had a similar attack, nor had she ever fainted in her life. After recovering consciousness from this attack, whose character is, of course, doubtful, she at once became generally weak, and forthwith went through the most severe paralytic seizure she has ever had. At 12 o'clock she was helpless. Her face was involved; she breathed only with the greatest difficulty, and had artificial respiration during the night. She was also given digitalis. The following day she was somewhat better, but still helpless. The next day, about forty-eight hours after the onset, she was able to get up, but remained very weak. She was not entirely strong for seven days. She had been married three weeks before this attack. During her wedding journey she had been travelling and walking much more than was her custom.

After this violent seizure she seemed to improve, and had milder attacks at longer intervals. This continued up to February, 1897. Then she had tonsillitis, and a serious attack, but less so than the one above described. This was followed by slight attacks up to a week ago (September, 1897), but for a month she has been feeling weak in the characteristic way. In September, 1897, a week before I saw her, she had another severe attack, coming on after a period of anxiety. She went to bed well, and woke up perfectly helpless, with both face and respiration affected; the attack lasted about twelve hours: four hours later she came to Boston. She states that her muscles always feel weak and sore after an attack.

Physically, she is an exceedingly strong and healthy looking woman. She has no disturbance in the bowel or menstrual functions, and, apart from her family infirmity, considers herself well. Owing to the relative infrequency of her attacks, she never lost work at school, and was the valedictorian of her class. She is a woman of considerable general culture.

Her pulse was 88; slightly irregular. She sleeps fairly, has no headaches, and is not of a nervous temperament. She has noticed no difference in the urinary secretion during and between the attacks.

This patient has not been seen since this visit in September, 1897. I have only learned from other members of the family that she is, on the whole, improving, but by no means free from the attacks. In average they are less severe than in Case I., although occasionally very much more so, even threatening a paralysis of respiration, as already described.

The two cases are evidently identical in their essential nature, though differing somewhat in detail. The examination

has been in neither case, notably in the second, as complete as desirable, but amply sufficient to establish the diagnosis, and add, we hope, some points of interest to the literature already collected.

In consideration of the relative fewness of the cases reported, and extreme rarity and growing interest of conditions of this character, we offer the following abstract of cases hitherto appearing in literature, in the order of their publication:

Case I. Cavaré: Woman, 24 years old, several attacks of a general paralysis, lasting five to eight hours; quotidian type; relieved by quinine; no statement as to malaria.

Case II. Romberg: Case of paralysis of lower extremities, occurring in three attacks of the quotidian type. Disappeared on administration of quinine. Thought by Romberg to be due to intermittent fever (malaria).

Case III. Hartwig (quoted by Westphal): Male, 23 years old; healthy family; five years before had had tertian intermittent fever. Onset; "tired feeling" in legs; increased; spread to arms. On third day moved with great difficulty. Following night, paralyzed; legs, arms, body and movements of head. Facial muscles free. Speech, breathing, swallowing somewhat difficult. No incontinence. Sensibility unaffected; general condition good; no fever; some sweating; after twenty-four hours improvement in following order: neck, fingers, arms, body, legs. Similar attacks lasting about twenty-four hours. Attack later observed; muscles of respiration, except diaphragm, affected; coughing and sneezing impossible. Loss of reflexes. Electrical excitability almost abolished; at same time numbness, formication and cramp in paralyzed parts. Temperature normal. Paresis of extremities persisted and increased. Secondary contractures. Quinine merely temporary effect. Paresis increased by rest, diminished by movement. Patient lost fifteen pounds. (Later history not given.)

Case IV. Samuelsohn: Male, 38 years old; affection began at 18. At first, two attacks yearly; later one a week. Legs, body fully paralyzed; upper extremities only partially. Sensation undisturbed. In ten hours recovery. Quinine and other drugs unavailing. No electrical tests made, nor observations on the reflexes. Samuelsohn thought case of hysterical character.

Case V. Schachnowitsch: Father of patient similarly affected. Said to have died from increasing attacks. One brother epileptic. Patient, a man; attacks for twenty-five years; usually begin at night; also has abortive attacks indicated by subjective sensory disturbance and weakness; attacks could be checked by energetic movements; muscles tense.

Case VI. Gibney: Child of 7; after malarial attack, paralysis of four extremities. Sensory disturbances. Slow recovery, after months. Muscular atrophy. (A doubtful case.)

Case VII. Gibney: Child of 6; similar to preceding; slowly recovering paralysis; much constitutional disturbance; malarial. (A doubtful case.)

Case VIII. Westphal: Boy of 12; strong. Onset; weakness in legs with a pricking sensation. Great thirst and desire to urinate. Some minutes before he could pass urine. Three hours later could not stand. Slight movements of hips and arms possible; at midnight complete paralysis. Seen by Oppenheim at 4 A. M. Mind clear, cranial nerves free. Turning of head difficult. Coughing, sneezing

impossible. Complete flaccid paralysis of arms and legs; sensibility normal. Plantar reflex lacking; cremaster and abdominal present; knee-jerk weak and not always obtainable. No dyspnoea.

Electrical examination: Faradism; strong, painful currents, very weak response from nerves. None from left peroneal nerve and its muscles. At 8 A. M. gradual return of reaction to G. and F., direct and indirect. Quick contractions when present at all.

Improvement: in evening could walk. No enlargement of spleen. Sweating. Temperature 37.7. Recovery on the following day, except for slight electrical changes in certain muscles. Similar attacks followed. Movement prevents onset of attack.

Etiology: No heredity. Scarlet fever followed some years later by unknown illness. Four weeks after exposure to draught sudden paralysis. Well between attacks which were at first four to six weeks apart, and lasted about twenty-four hours. Same patient studied again later by Oppenheim. Condition between unchanged. Attacks usually at night; awakes paralyzed, decreasing in intensity until following evening. Arms, legs, head paralyzed; face, tongue, larynx and eye muscles free. Thirst, heat, sweating. No excretion of urine. Intensity of paralysis not always the same. No pain; no disturbance of consciousness; no paræsthesia, except tickling in soles of feet. Swallowing practically unhindered, knee-jerks lost. Heart, slight temporary enlargement, and murmurs indicative of insufficiency. Heart normal between attacks. Electrical examination; results not uniform. In general, complete loss in certain muscle groups at height of attack. During attack often increased skin resistance. Later observations; slight atrophy of those muscles, with poorest electrical reaction between the attacks. Permanent weakness of legs.

Case IX. Fischl: Girl, 8 years old, good heredity. November, 1884, scarlatina, nephritis; complete recovery. Following May, lassitude, headache and backache, appetite poor. Somnolence, difficulty in rousing her from sleep. One morning could not stand or sit without support. Examination, mind clear; anxious expression; somewhat rapid pulse; temperature of head, body, arms normal; of legs and lower part of thighs ice cold, and remarkably pale; movements of head free. Cranial nerves free, movements of body good. Sitting up, or rising from prone position not possible. Arms normal when examined. Said to have been weak at first. Lower extremities completely paralyzed; flaccid. Sensation, diminished in affected areas. Abdominal reflexes present. Knee-jerks lost. Plantar reduced. No muscle excitability. No splenic enlargement; internal organs normal; urine and faeces normal, and normally passed. Attack lasted three hours. Sudden and complete recovery. Second attack, a few days later, also in the morning. Electrical examination; faradism; arms, normal; legs, no reaction to strong currents, excepting some muscular contraction in anterior crural distribution. Duration, five hours. Slower recovery. Normal reactions between attacks to galvanism. Third attack, less than an hour's duration. Same otherwise, as before. Fourth attack, length one half hour during day; arms weak, then legs. Recovery of arms first. *Prodromata:* headache, pain in back and legs, yawning, weakness. Later attack, twenty-four hours' duration, right foot only affected. Subsequent attacks irregular, characterized by drowsiness, one quarter to one half hour long, usually in afternoon. Child slept normally except for occasional stretching. Later, feeling of being tired. Subsequently became entirely normal (somewhat doubtful case).

Case X. Cousot: Mother and four children; no heredity otherwise. All strong, but smaller children of family of ten, affected. The mother often lost motor power at times twice weekly, through life.

Son, man now of 34; attacks began at 14, increased up to 20, then stationary. Strong in appearance; perfectly healthy on examination between attacks; attacks very frequent; usually began at night. First, feebleness of articulation; desire to walk; no sensory disturbance; usually arms last to lose power and first to regain it. Emotions increase tendency to attacks. Work and fatigue make them more violent. Duration, eight to ten hours; most helpless at third or fourth hour of attack. Muscles of face always spared. Deglutition and speech difficult. Attacks deferred by exercise. Intelligence unimpaired. Sweating during attacks. No urinary nor faecal incontinence. Urine: no sugar, no albumin, much uric acid. Electrical reactions lost; galvanic response first appears on recovery.

The next Cousot case, not carefully observed; boy, onset at ninth year, similar to foregoing but more severe.

The other two cases, girls, onset in both about tenth year; precisely similar attacks.

Case XI. Griedenberg: Soldier; 22 years old; healthy; no heredity; similar attacks to foregoing; occasionally attacks limited to legs; cranial nerves free; certain muscles contracted.

Case XII. Pulawski: Man, 21 years old. Nothing significant in past history. Had had two previous short attacks. Present attack: went to bed well, at one A. M. could not move. No pain, sphincters unaffected. Examined in hospital; movements of arms not possible; slight movements of fingers. Same condition in legs. Could not sit up; lateral movements of head possible, not forward and backward. Muscles flaccid; respiration free, but superficial. Deep respirations and coughing impossible. Cranial nerves free. Sensation and muscle sense, normal. Knee-jerks lost. Cutaneous reflexes normal. Internal organs normal. Feeble faradic reaction. Hypnotism failed twice. No evidence of an antecedent intoxication. Urine; alkaline; specific gravity, 1017; no albumin, no sugar, abundant sediment of ammonium urate. Attack lasted one day; quinine given; following night much sweating, recovery.

Case XIII. Goldflam: Eleven cases in patient's family, on mother's side. No other neurotic heredity. Transmitted through both males and females. Onset usually between fifteen and twenty. Frequency varying from weekly to yearly attacks. Mother of patient had one attack in thirty-sixth year. Attacks generally more frequent in youth, but never cease altogether; in only one case was paralysis limited to legs.

Case reported in detail: Boy of 17. Previous history, typhoid fever, scarlet fever, with ear involvement, eczema; otherwise well. At thirteen paralysis of body, head, and extremities; duration three days. Saliva could not be swallowed. Following summer two equally severe attacks; later others, not so severe but oftener, lasting from twenty-four to forty-eight hours; then more severe, followed again by milder seizures. Attacks usually Friday, beginning at 6 or 7 P. M. First, weakness of legs, arms less affected, on well evenings much trouble with itching; none on evening of attack. No pain, chill nor fever. Constipation; complete paralysis, sparing head; thirst; sleepy during attack; consciousness otherwise undisturbed. Sense organs, speech, bladder, normal. Slight sweating during attack, more profuse toward its end. This, with acute itching for an hour ushers in end of attack. Return of movement in following order: Fingers, arms, body, legs, within a few hours. Capricious as to food, but no symptoms of intestinal disturbance. Drinks tea only on the evening before, and during attack. Physical examination: Flaccid paralysis, knee-jerks absent, plantar, abdominal and cremaster present. Sensation normal. No pain on pressure over nerves or muscles. No diminution

in amount of urine during attack. No enlargement of spleen. Electrical examination: faradism; quantitative diminution in nerves of arms; loss in legs. No reaction from muscles in either arms or legs. Reaction of facial nerve normal. Mechanical muscular irritability lost. After attack, nerve and muscle irritability normal, excepting small muscles of thumb; slow contraction; An C=Ca C; R. D. chiefly in small muscles of hands.

Further study of above case made four years later: Itching less prominent than before. Prodromata now; feeling of coldness in legs, and then in arms. Occasional thirst; attacks usually at night. Abortive attacks, certain groups of muscles affected. Attacks occasionally aborted by active exercise, usually unavailing. In irregular attacks, relapses in stage of improvement. At times arhythmic heart; systolic murmur at base. Slight accentuation of second tone; no enlargement; slow pulse; faint first tone in attacks.

Case XIV. Goldflam: Older brother of the foregoing; first attack at eighteen; attacks in general, similar; in one, dangerous asphyxia. Good muscular development. Electrical examination: direct and indirect irritability markedly diminished to both streams between attacks. Rapid fatigue of muscles. Slow contractions; polar changes. Ca C Te. obtained with weak currents. Faradic R. D. and partial R. D. In free intervals qualitative changes prominent; in attacks, quantitative. Loss in attack proportional to amount of paralysis. Mechanical irritability lost in complete attack. These changes in general found in both brothers. Goldflam mentions in a footnote to his article, to which reference has been made, two other cases in children, not yet adequately observed.

Case XV. Burr: Male of 30, no heredity whatever; no malaria. Began at 10. One attack about every four months since. Attacks last from one day to a week. Recovery gradual. Cranial nerves free. Slight involvement of neck muscles. Sensation, sphincters, mind normal. Attacks increasing somewhat in frequency. When seen by Burr, hemiparesis. Knee-jerk absent on left; present on right, but slight. Spleen not enlarged. No pain. Attacks always come at night; at times momentarily weak in the street, never at home. No electrical examination made.

Case XVI. Hirsch: Male of 26; no heredity, except mother had similar attacks, often of very quick recovery and with thirst during attacks. Patient previously well; first attack at 19th to 20th year; arms first heavy and stiff, then legs; obliged to go to bed. Paralysis not extreme. Well in twenty-four hours. One such attack yearly thereafter until 1892. Prodromata of heavy and tired feelings in legs for days before actual attack. In 1894 first decided paralytic seizure. Arms, legs, body, face, free. Duration, twenty-four hours; sudden recovery; thirst. Physical examination at hospital: mind clear; intelligence and speech unaffected. Facial and other cranial nerves free. Neck muscles markedly affected. Breathing superficial. Paralyzes inspiratory and expiratory muscles, excepting diaphragm. Lungs normal. Heart dullness enlarged; first sound at apex impure, slight accentuation of second pulmonic. Insufficiency of mitral valve. Pulse regular. 78: abdominal muscles movable. Spleen normal. Complete paralysis of arms and legs; deep reflexes lost, also superficial reflexes, excepting abdominal, which were weak. Idio-muscular irritability of paralyzed muscles weaker than normal. Sensibility normal; also muscle sense. Occasional sharp pains in left foot; no painful points. Bladder and rectal functions normal. No albumin, no sugar, no increased sweating; normal temperature, good appetite. No electrical examination made. Rapid recovery during night; entirely recovered following morning; heart practically normal. Attack twenty-four hours coming on, forty

hours' duration. Cause of attack as given by patient, "drinking of tea or grog the night before."

Case XVII. Rich: 22 cases occurring in five generations, no individual case particularly described. The type is as follows: Family affection, no other neuroses or psychoses in the family history. Cause, exposure to cold. Paralysis of tonic character following and affecting various groups of muscles, preferably the face. On one occasion complete motor paralysis, excepting tongue, from sleeping in moist underclothing. Paralysis of tongue may be brought about by holding snow in the mouth, taste remaining uninvolved. Sensation in general unaffected, mind clear. Desire to urinate; sphincters normal; complete temporary recovery with considerable antecedent weakness. The affection has never skipped a generation. No mention made of electrical changes, nor of reflexes during, or between the attacks (doubtful cases, belong rather to the congenital myotonias).

Case XVIII. Bernhardt: Man; affection began in extreme youth; sudden attack of general weakness. Difficulty always in walking; tendency to stumble. Some muscles hypertrophic. Three years later general weakness; electrical reactions diminished; mind clear (doubtful case).

Case XIX. Bernhardt: Son of preceding case; almost daily attacks. Tendency often to cough, but not possible; cyanosis, threatened suffocation, sweating. Father similarly affected. Examination three years later: Attacks on waking, going to bed well. Immovable; sweating; no loss of consciousness. Speech at times slightly affected. Two years later unstable, but special senses normal. Slight quantitative diminution to electrical current. Knee-jerk present; walks with great difficulty. No difficulty in rising from seated position. Bladder and rectal functions normal. No albumin, no sugar. Attacks similar, but less frequent than formerly. Never come on when exercising, only after rest. Neck muscles affected, cranial nerves free. Attacks last about fifteen minutes (somewhat doubtful case).

Case XX. Goldflam (reported in 1897): Man of 22; attacks began in eighth year, and have increased in frequency. Attack usually begins in the evening with weakness of arms and general feeling of exhaustion. Sleep usually follows; on waking, often complete paralysis. Duration, twenty-four to forty-eight hours. Mind, speech, deglutition, sphincters, free. Recovery begins in evening. At times attacks sudden in onset. Attacks much more frequent in summer than in winter. Muscles well developed apparently, but strength diminished in free intervals; also changes in electrical excitability, including muscles of face. Examination during an attack showed usual conditions; toward end of attack, urine had traces of albumin.

Case XXI. Goldflam: Boy of $7\frac{1}{2}$; brother of previous case. First attack in fifth year, examination incomplete; certain electrical changes in free interval demonstrated.

Case XXII. Goldflam: Man of 28; first attack in eleventh year. Infrequent attacks of from twenty-four to seventy-two hours' duration. Attacks at night. Extremities, body and neck muscles paralyzed. Coughing difficult, appetite poor, mind and sphincters unaffected. Marked electrical changes; slow contraction.

Case XXIII. Goldflam: Woman of 25, sister of previous case. Onset of attacks at twenty-four. Hands, arms, legs affected, in order named. Paræsthesia in fingers. At times complete paralysis. Intelligence, speech, sensibility, sphincters, unaffected. Loss of appetite; recovery first in arms, followed by head and legs. Diminution to complete loss of reflexes during attack, with marked quantitative diminution in faradic excitability. Pulse irregular, rapid; heart mur-

mur; no enlargement of heart. Trace of albumin in urine during height of attack.

Cases XXIV and XXV. Taylor: Complete report already given.

(To be continued.)

219. SYNDROME SYRINGOMYELIQUE AVEC HEMIATROPHIE FACIALE ET TROUBLES OCULO-PUPILLAIRES (The Syndrome of Syringomyelia with Hemiatrophy of the Face and Oculo-Pupillary Disturbance).

Queyrat et Chretein (La Presse Médicale, December 24th, 1897.

The patient had had in infancy a series of abscesses of the skin. He did not walk until three years of age, but otherwise appeared well, and displayed normal intelligence. At the age of eighteen years he had a very painful panaritis upon the index finger of the right hand, causing the loss of the terminal phalynx. Three years later he had a second panaritis upon the left thumb, much less painful, but causing the same mutilation. At this time there was some loss of power in the left arm and a succession of panaritides, and he was treated by various physicians for leprosy without any improvement. His condition in 1896 was as follows: There was kyphosis and slight scoliosis in the dorso-cervical region, mutilation of the thumb, index and middle fingers of the left and of the thumb and index finger of the right hand. Some very large cicatrices, representing the situations of the infantile abscesses were found upon the arms. The face was markedly asymmetrical, the left side being distinctly smaller than the right, but there were no motor disturbances other than those produced by the difference in size. The hair grew equally well on both sides. There was slight paresis, chiefly in the arms. The right hand was somewhat weaker than the left. Reactions of degeneration were present in the muscles of the hands. Sensibility was diminished in the left half of the face, the left shoulder and the left arm and hand, most markedly in the latter situation, and involved touch, pain and temperature. The tactile sensibility was preserved in the right forearm, but the pain and heat senses were lost. Both the pupils were myotic, reacting well to light, but poorly to accommodation. The visual field was normal; the patient, however, was color blind, not being able to perceive green. The case is interesting on account of the existence of facial atrophy and the possibility of leprosy, although a careful search for the microörganisms in an excised piece of skin was negative.

SAILER.

EXPERIMENTAL RESEARCHES ON THE LOCALIZATION OF THE SYMPATHETIC NERVE IN THE SPINAL CORD AND BRAIN, AND CONTRIBUTIONS TO ITS PHYSIOLOGY.¹

(ABSTRACT.)

By B. ONUF (ONUFROWICZ), M. D.,

Associate in Pathology, Pathological Institute of the New York State Hospitals;
Lecturer on Nervous and Mental Diseases in the New York
Polyclinic.

AND

JOSEPH COLLINS, M. D.,

Professor of Diseases of the Mind and Nervous System in the New York Post-Graduate
Medical School; Visiting Physician to the City Hospital; Neurologist
to the Post-Graduate Hospital, St. Mark's Hospital,
and to the St. John's Guild Hospital.

The anatomy and physiology of the nervous system have long been favorite subjects for the study and speculation of scientists. In latter years the method of metal impregnation, inaugurated by Golgi, and the methylene blue method, first successfully utilized by Ehrlich, have lent themselves to the study of this obscure part of the body, and many investigators, of whom we may mention Kölliker, His, Ramon y Cajal, Van Gehuchten, Retzius, Dogiel and Sala, have illumined our knowledge of the minute structure and of the architecture of a large part of the nervous system. Thus far, however, few authors have undertaken to determine the manner in which the sympathetic is connected with, or, better said, localized in, the spinal cord and brain. Of these few, some have speculated from clinical data alone regarding such localization. According to Mott, for instance, Ross was the first to suggest that in tabes the visceral crises and other disturbances of a similar nature are due to affection of the

¹ Read before the American Neurological Association, May 27th, 1898. From the Pathological Institute of the New York State Hospitals.

cells of Clarke's columns. Sachs says "it is not a great stretch of imagination to suppose that tactile sensation and the sensory impulses by which reflex action is excited pass through the lateral series of fibres, whereas those fibres connecting with the columns of Clarke in all probability have to do with the functions of coördination and with the transmission of visceral sensations." The merit of the first attempt to study in a systematic manner the distribution of the "visceral" nerves in the brain and spinal cord is due decidedly to Gaskell. To enter into the details of the highly ingenious plan on which his researches were conducted would lead too far. We can only hint at some of the principal points. Gaskell had demonstrated that in the nerve roots of the cerebrospinal nerves certain medullated fibres distinguish themselves by the fineness of their calibre. He had shown in a convincing manner that these fine fibres represented the visceral fibres of the roots. Furthermore, he had demonstrated the presence of these fine medullated fibres in many of the rami communicantes. By following the course of these fine fibres in the spinal cord he came to definite conclusions, the most important of which are: (1) That the visceral nerves become connected in the spinal cord with the large cells of the lateral horn, which he considers to be a nucleus for efferent fibres to striated splanchnic muscles; (2) that the cells of Clarke's columns give origin to inhibitory fibres for the splanchnic glandular system and the muscles of the viscera and vascular system; (3) that the solitary cells of the posterior horns furnish a motor supply for the muscles of the viscera, and finally (4) that the small cells of the lateral horn form a nucleus of katabolic (motor) nerves to the splanchnic glandular system and to the muscles of the vascular system.

There can be no doubt that some of the premises on which Gaskell bases his conclusions are, in part at least, erroneous. It is an erroneous statement, for instance, that the vasoconstrictor nerves arise only in the thoracic

region of the spinal cord. Yet we shall see that, in the main, Gaskell has hit very near to the mark in most of his final conclusions, and we can only praise his ingenious researches, and recommend them for close study to him who wishes information of the structure and central distribution of the sympathetic nervous system.

Mott contends against Gaskell's view that the axis cylinder processes of the cells of Clarke's columns become fibres of the anterior roots, claiming justly that these axis cylinders are continued as fibres of the direct cerebellar tract. Mott considers the nuclei of the funiculi cuneati and Deiters' nucleus to be the homologue of Clarke's column in the medulla oblongata. Blumenau had previously reached the conclusion that the large cells in the lateral portions of the funiculus gracilis and (chiefly) funiculus cuneatus were the homologues of Clarke's columns. Mott's further views regarding the connections of the sympathetic nervous system with the cerebrospinal axis may be summed up as follows:

"The fine, centrifugal, splanchnic fibres which Gaskell found in the anterior roots originate from the bipolar cells of the tractus intermedio-lateralis (lateral horn) and from the solitary cells of the posterior horn. The vago-glossopharyngeal nucleus (the one situated beneath the floor of the 4th ventricle) is to be considered as the continuation of the tractus intermedio-lateralis, having the same physiological significance in the medulla oblongata as the latter has in the cord. Other larger cells of the tractus intermedio-lateralis have altogether other functions, and are, perhaps, related to the antero-lateral tract."

Biedl cut the splanchnic nerves in dogs and studied the ascending degeneration in the spinal cord. His conclusions are formulated in a rather vague manner, so that it is difficult to gather where he conceives the location of the centres of the efferent fibres to be, and where he believes the afferent fibres to end in the spinal cord. Yet it would seem that he found a splanchnic motor centre

in the lateral horn of the lower cervical and upper dorsal regions. We must not forget to add, however, that the purpose of Biedl's researches was not so much to establish the localization of the splanchnic nerve in the spinal cord as it was to study the histological *character* of the spinal cell changes occurring after section of the nerves mentioned.

Aside from the investigations just discussed (Gaskell's, Mott's and Biedl's), we have found no literature relating to the localization of the sympathetic or visceral nerves in the spinal cord or brain.

In view of the fact, then, that so few investigators have attacked this subject, and particularly that the conclusions of these are disharmonious, it seemed to us justifiable to retake a new experimental and critical survey of the subject.

Notwithstanding the liability to erroneous conclusions by homologizing results obtained from experimentation on animals, we decided to adopt the experimental method for our investigations. It was originally intended to confine our researches to the allocation of the sympathetic nerves in the spinal cord and brain, but some of the physiological observations which the experiments permitted us to make proved interesting enough to be embodied in the report of our findings. In this connection it is necessary to state that the results of our investigations are embodied in a monograph on the sympathetic nervous system, which will soon be published in the Archives of Neurology and Psychopathology. Here we shall attempt to give only a brief abstract of our conclusions, and to illustrate our principal findings by diagrams and drawings.

Our mode of procedure consisted in extirpating various parts of the nervous system, and studying the consecutive ascending degeneration in the spinal cord and oblongata. In all, eight cats were thus successively operated upon. The following operations were performed:

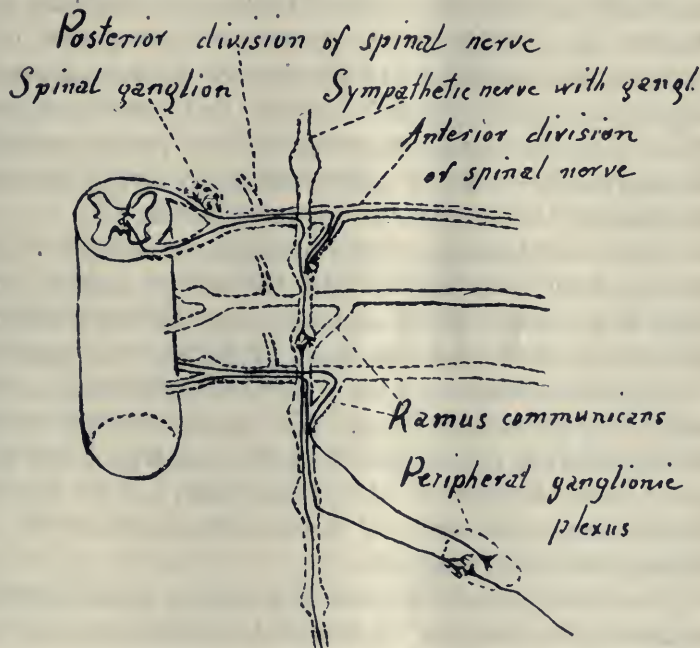
1. Extirpation of the stellate ganglion; a ganglion that corresponds to the cervical and the first thoracic

sympathetic ganglion of man fused into one common ganglion. This operation was done in three young cats.

2. Extirpation of a piece of the thoracic sympathetic nerve with three ganglia in two young cats.

3. Extirpation of the lumbar sympathetic nerve with three ganglia in one young cat.

FIG. I.



4. In two young cats the semilunar ganglion, which is known as the abdominal brain, was extirpated.

Although it is difficult to encompass in a few paragraphs the results of our experiments and observations, we shall endeavor to state the more important conclusions. These can be classified into: First, localizatory; second, physiological; third, general physiological remarks.

I. Localizatory: Our conclusions concerning localization may be summed up as follows:

Most of the afferent (sensory) fibres of the sympathetic nerves do not originate from cells of the spinal ganglia, as

Kölliker claims; on the contrary, they must have, in accordance with Dogiel's view, their cells of origin within the ganglia or plexuses of the sympathetic system.

We believe that the *efferent* fibres of the sympathetic take their origin from the cells of the following groups: First, the paracentral group; second, the small cells of the lateral horn, and third, probably also the small cells of the intermediate zone. By way of explanation, we may say that we have designated as paracentral group that collection of cells situated to both sides of the central canal, directly ventrad of Clarke's column, and sometimes confluent with the latter, especially in very young animals. By intermediate zone we understand an area lying between the bases of the posterior and anterior horns.

The *afferent* fibres of the sympathetic are connected by their terminal arborizations with the cells of Clarke's column, and it seems quite probable that the large cells of the intermediate zone, especially of Bechterew's nucleus, bear the same relation to the visceral afferent fibres as the cells of the vesicular column. We concede that the whole zone separating the anterior from the posterior horns has relations to the fibres of the sympathetic, but we do not thereby imply that many of the cells therein have not altogether different functions.

We saw vertical fibre bundles emerge from Clarke's columns, and bend off in horizontal (dorso-ventral) direction; part of them seemed to lose themselves in what we call the paracentral field. These fibres we have much reason to consider either as direct afferent fibres of the posterior roots or as collaterals. In our monograph we give detailed arguments in favor of the view that they terminate around the small cells of the paracentral group (perhaps also of the intermediate zone), and are thus destined for the enactment of spinal reflexes in the domain of the vegetative nervous system.

In young animals (cats) Clarke's column and the paracentral cell group coalesce almost into one group. Prob-

ably in the adult the separation is also incomplete, so that the two may have partially common functions in such manner that part of the cells of Clarke's column (the larger ones) are concerned in afferent, the other (the smaller ones) in efferent functions. Similarly, the large sporadic cells that one meets in the paracentral group may have afferent, while the smaller ones, which form the chief contingent of the group have efferent functions.

Two weeks after extirpation of the 3d, 4th and 5th lumbar sympathetic ganglia we observed degenerative changes, both in the cells of Clarke's columns and in the fibres passing into them from the posterior roots. The degeneration in the fibres reaches from the 3d lumbar up to the 13th dorsal segment; on the other hand, the inferior (caudal) limit of the cell changes must be looked for in the 1st lumbar segment, showing that the cell changes occupy, on the whole, a higher level than the fibre changes, that, accordingly, the afferent fibres of the lumbar sympathetic nerves, entering the spinal cord by way of the posterior roots, make, after having arrived at Clarke's columns, a longitudinal course cephalad to terminate around cells of a considerably higher level.

From the distribution of the secondary atrophies observed in the spinal cord four weeks, and six months after extirpation in one case of the 7th (or 6th) to 9th, in the other of the 7th to 11th, thoracic sympathetic ganglia in young cats, we conclude that, on the whole, the fibres—at least the afferent, probably also the efferent—coming from the ganglia of the lower half of the thoracic sympathetic cord, take a rather horizontal course in the spinal cord, becoming connected with spinal cells of the same level, but that part of these fibres descend through the distance of one or more segments before reaching the cells around which they terminate (or from which they originate if they be efferent fibres).

Extirpation of the stellate ganglion causes within a few months retrogressive changes of an atrophic order

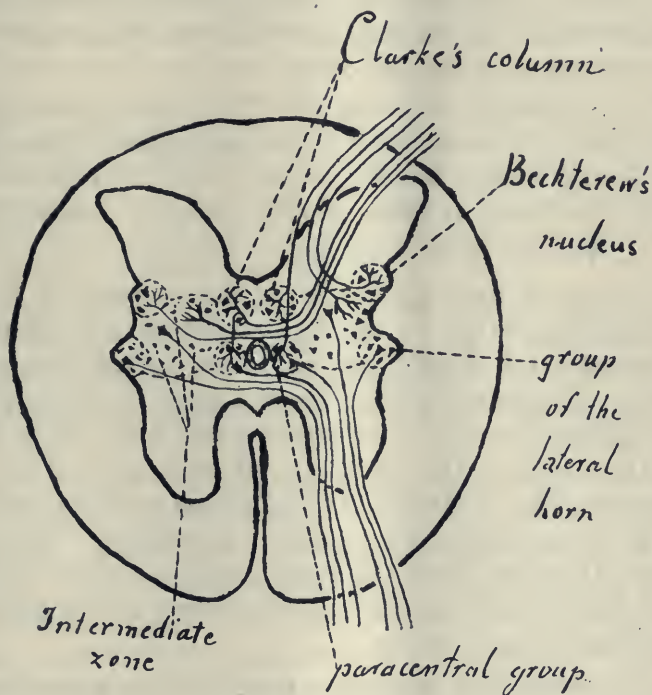
in the cells of both lateral horns, of both paracentral groups and of both columns of Clarke. These changes extend downward at least to the 9th dorsal segment, showing that many of the afferent and also of the efferent fibres from the stellate ganglion make a long descent in the cord, or possibly in the sympathetic nerve, becoming connected partly with the same cells with which the fibres from the lower portion of the sympathetic cord form connections. We may conclude that the afferent fibres of the sympathetic system, after T-shaped division, become ascending and descending, and thus become connected with several levels of Clarke's columns simultaneously.

Regarding the function of the paracentral group, we have adduced arguments in favor of the view that it may be concerned in vascular and in visceral motor innervation. Clarke's column, besides being a terminal station for afferent fibres conveying impulses from the vegetative organs, may be instrumental also in conducting sensory stimuli from the muscles, tendons, joints and bones to the cerebellum, being thus largely concerned in functions of equilibrium. Stilling's sacral nucleus, situated in the 3d sacral segment, is possibly a coalesced Clarke's column and paracentral group.

Regarding the representation of the sympathetic in the oblongata, we find that it has not yet been proven that the vago-glosso-pharyngeal nucleus situated beneath the floor of the fourth ventricle is a terminal nucleus of purely sensory or afferent function. We are much inclined to share Forel's and Gaskell's view that the nucleus is, on the contrary, predominantly motor in such sense that the axis cylinders of its cells become efferent fibres of the IX. and X., probably also partly of the XI. nerve. The fact, however, that by extirpation of the stellate ganglion—aside from afferent fibres—*only visceral* (vegetative) *efferent* fibres and *no somatic motor* fibres of the vagus nerve (which give off a strong communicating branch to the ganglion), become interrupted, in connection with

the observation that as a secondary consequence of such lesion the spinal division of the vago-glosso-pharyngeal nucleus undergoes some, although very slight, atrophy, while the nucleus ambiguus remains normal, leads us to conceive furthermore that the vago-glosso-pharyngeal nucleus gives origin only to visceral (vegetative) efferent

FIG. II.



fibres of the vago-glosso-pharyngeal, and in part also the accessory nerve, and that the nucleus ambiguus gives origin only to somatic efferent fibres of these nerves, that is, to motor fibres supplying striated muscles. In other words, in relation to the so-called lateral mixed system of nerves (which includes the IX., X. and XI. nerves) the so-called vago-glosso-pharyngeal nucleus is probably the visceral (vegetative), the nucleus ambiguus, the somatic nucleus.

The so-called vago-glosso-pharyngeal nucleus is, furthermore, probably the homologue of the paracentral group. The homologue of Clarke's column we believe to be a nucleus accompanying the solitary bundle at its ventro-lateral border. The relation of the afferent fibres of the lateral mixed system (IX., X. and partly XI. nerves) to the two nuclei just mentioned is probably such as we have tried to demonstrate as existing between the spinal visceral fibres on one side and Clarke's column and the paracentral group on the other.

In accordance with this view, the vagus fibres which have been seen terminating in the vago-glosso-pharyngeal nucleus by Van Gehuchten, Kölliker, His and others are to be considered as afferent reflex fibres or collaterals.

II. Physiological conclusions:

In regard to the influence of the sympathetic upon lachrymal secretion, our results were rather contradictory. Removal of the stellate ganglion in one animal apparently prevented secretion of the lachrymal gland of the operated side when pilocarpine was instilled, while in two other cats, on the contrary, the secretion was more profuse on the operated side. Naturally, the lachrymal secretion was an artificial one caused by a poison, pilocarpine. We conclude, therefore, that the results were so contradictory that further experimentation is necessary before positive conclusions can be drawn.

In reference to the sweat secretion, our experiments seemed to warrant the assertion that not all sweat secretory fibres of the forepaw pass through the stellate ganglion, and through the main trunk of the sympathetic in general, as Luchsinger and Langley assume, but that a good portion of them follow other pathways, and that these fibres develop a compensatory function so strongly as to entirely mask the loss of function. But yet we had to note the paradoxical fact that in a cat in which the stellate ganglion was removed, there was sweating of

all the paws except the left forepaw, as the result of the animal's struggles during etherization.

In reference to the influence of the sympathetic system on the pupil, our experiments led us to believe that the cervical sympathetic contains not only pupil-dilating fibres, but very probably pupil-contracting fibres as well.

Regarding digestion, we found that disturbance of that function followed invariably on removal of the stellate ganglion, of the lower thoracic portion of the sympathetic and of a semilunar ganglion. The digestive disturbances that ensue after removal of the stellate ganglion are, however, more marked and more persistent than those noted after removal of the lower thoracic sympathetic. They consisted of diarrhoea and of putrefaction of the fæces. They were more or less remote symptoms, and they showed a progressive tendency.

We observed that removal of one stellate ganglion, as well as defect of the lower part of the thoracic sympathetic (including the splanchnic at this level), gives rise to attacks of sneezing, to paroxysms of coughing and to hiccough. The cough occurs not only spontaneously, but a paroxysm of coughing could always be precipitated by stroking the animal's back, particularly the nuchal portion. Removal of the stellate ganglion causes, in addition, first a mucous, then a purulent discharge from the nasal mucous membrane. In one case it produced a chronic purulent bronchial catarrh with lobular infiltration of the lungs. The attacks of cough and hiccough gave the impression of nervous symptoms due to defective inhibitory action. The respiratory disturbances were more grave in a case of removal of the stellate ganglion than in a case in which resection of the thoracic sympathetic in its lower portion was done. We noted that resection of the lower part of the sympathetic was followed by diabetes, and, considering the large amount of sugar found four months after the operation, we are led to the belief that the glycosuria caused by such lesions is not tem-

porary, but permanent, and seems to have a tendency to increase rather than to diminish.

In reference to the effect of extirpation of the stellate ganglion on the local temperature, we found that there was an immediate and a remote increase of from one to two degrees Fahrenheit.

Concerning the pilomotor nerves, we concluded that, although they have, on the whole, the segmental distribution which Langley and Sherrington attribute to them, there must be a collateral supply, or a direct cerebrospinal supply, which can, in the course of time, entirely replace the functional loss which extirpation of three or four successive ganglia causes.

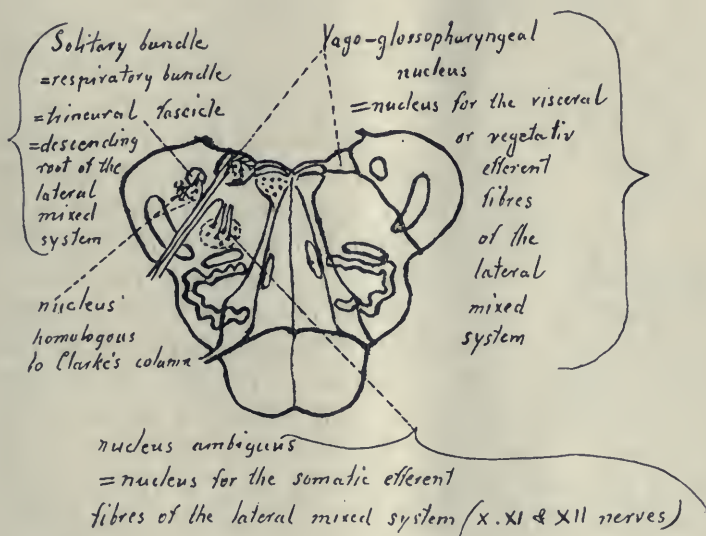
The trophic influences which we observed in connection with lesions of the sympathetic were most evident after removal of the stellate and the lower thoracic ganglia. They were bilateral, although quite irregular in distribution, and were predominantly cutaneous (partial alopecia). It is probable that the nasal, bronchial and laryngeal secretion already spoken of may be on a trophic basis.

III. General Physiological Remarks:

The essential influence which the sympathetic system exercises on the vegetative life of the organism has been amply demonstrated by numerous physiological observations. Inasmuch as some vegetative functions are exquisitely vital, we may say also that the sympathetic system possesses in high degree vital function. This is confirmed by our observations. In very young cats lesions of the important parts of the sympathetic invariably proved fatal. Even if the animals outlived such operations as extirpation of the semilunar ganglion, or removal of the stellate ganglion, or resection of the lower part of the thoracic sympathetic, they invariably died, usually a few hours or days, afterward. One cat of four weeks of age survived the removal of one semilunar ganglion three weeks, being at first quite playful and apparently healthy, but at the end

of two weeks he was attacked by diarrhoea, and died in a state of collapse. Even a cat of five and a half weeks, in which we had removed three lumbar ganglia, would have died from collapse two weeks after the operation, had we not preferred to kill it by chloroform, and in this case no tangible cause of the collapse, except the defect of the said three ganglia, could be found.

FIG. III.



We desire to call attention to the fact that the death of many animals during the operations was caused by pulling upon the sympathetic nerve or bruising of a sympathetic ganglion. We noted that this was especially the case in operating to remove the stellate ganglion. Although the animal would be breathing vigorously and fully immediately before, as soon as the stellate ganglion was pulled upon, or as soon as its connection with the thoracic sympathetic nerve was severed, respiration became suddenly arrested, and the animal promptly died.

With older animals, that is, with cats which had reached the age of five or six weeks, we succeeded much better, and three of them lived from three to five months after the operation, when they were killed.

In closing, we wish to call attention to a method of physiological research which may serve to enlighten us on points for which the other methods give us no sufficient information. This method consists in studying, not the immediate, but the remote effects of injuries of certain loci of the nervous system; of investigating not only the perversion or loss of function, which is the immediate result of the removal or section of some ganglion or nerve, but also the compensation of the functional defect that occurs in the course of time. In this manner it is often possible to determine whether certain functions are performed exclusively by a definite nerve or ganglion, or whether other nerves or ganglia share in the fulfillment of this function. Illustrations of the truth of this are given in the observations made by us on the pupils of cats in which a stellate ganglion had been removed. The immediate consequence of this operation was reduction of the size of the pupil of the operated side to one-third, or less, of the size of the other pupil. Gradually, however, the difference in the size of the two pupils diminished, until in the course of from three to five months, it had entirely disappeared, showing in the most convincing way, by this compensation of function, that not all pupil-dilating fibres are derived from the cervical sympathetic nerve and stellate ganglion. The method mentioned has given another interesting result bearing on the same point. When, three months after the removal of one stellate ganglion, the ganglion of the other side was removed, a test of the pupillary reaction showed that the pupil of the side first operated upon contracted much more intensely and more rapidly to light than the other pupil. This fact can hardly be explained otherwise than by granting that the cervical sympathetic contains not only pupil-dilating but also pupil-

contracting fibres. Owing to this compensation, the pupil of the side on which the stellate ganglion had been removed three months previously to the test contracted much more promptly than the pupil of the other side, on which the ganglion had been extirpated just before the test.

No less interesting were the results which we obtained regarding the sweat fibres of the forepaw of the cat, and regarding the influence of the cervical sympathetic on lachrymal secretion. Twenty-five days after extirpation of the left stellate ganglion injection of one centigramme of pilocarpine caused no perceptible change in the state of the left forepaw, while when an injection or instillation of pilocarpine was made three or four and a half months after this operation (in two other cats), the forepaw of the operated side sweated quite abundantly, and in one case apparently no less than that of the other side.

Moreover, injection of pilocarpine three weeks after extirpation of the left stellate ganglion caused profound lachrymal secretion on the healthy side, the eye of the operated side remaining dry; while, on the contrary, three months after this operation (in another cat) the eye of the operated side secreted much less than that of the healthy side when pilocarpine was injected. In a third animal, finally, four and a half months after the defect of the ganglion, pilocarpine instillation produced lachrymal secretion of both eyes in an equal degree.

The contrast between the direct and the remote consequences of the defect of certain parts of the sympathetic system is further shown in quite an opposite direction. While such defects seem at first not to cause any disturbance of certain functions, such disturbances often make their appearance weeks, and even months, after the defect is created and show a tendency to progression. No legitimate conclusions could be drawn as to the effect of the removal of the stellate ganglion upon the gastric and intestinal functions during the first four weeks

after such removal, because during this period these functions appeared quite normal. Nevertheless, they became markedly disordered later. In the same manner two cats which were deprived of the semilunar ganglion showed no symptoms in the first two weeks after the operation, but at the end of that time one of them was taken with diarrhoea, and finally, three weeks after the operation, it died in a state of collapse. The second cat did not begin to have vomiting attacks until three weeks after the injury had been inflicted.

In like manner the disturbances of respiration observed after removal of the stellate ganglion, or the lower portion of the thoracic sympathetic nerve, differed in their immediate and remote consequences. In one case, for instance, pertussis-like paroxysms set in as late as two months after resection of the thoracic sympathetic nerve with the adjoining piece of the splanchnic.

The clinical importance of these facts needs no mention.

DISCUSSION.

Dr. William G. Spiller said that the paper read by Drs. Onuf and Collins contained so much valuable material, and was of such a character, that in discussing it one could hardly do justice to it after hearing it read once. The importance attached by the authors to the columns of Clarke and the cells in this region seemed to be justified by the investigations of others. Marinesco has advanced the view that Morvan's disease may be due to an affection of the posterior horns and intermediate gray matter, and the speaker said that about two years ago he, in connection with Dr. Dercum, reported a case of syringomyelia with arthropathy of the shoulder joint, in which the lesion in the cervical cord was limited to the posterior horn on the same side as the arthropathy. Dr. Spiller said he was inclined to believe that the cells of the intermediate gray matter, between the anterior and posterior horns, may be concerned with vasomotor and similar functions.

Drs. Onuf and Collins spoke of the presence of pupillary fibres in the sympathetic; the investigations of Madame Dejerine, Oppenheim, and others have fully established the fact that these fibres leave the spinal cord through the upper thoracic roots. The statement made by Onuf and Collins that

constricting fibres of the pupil are contained in the sympathetic is of much interest.

Marinesco found the posterior nucleus of the vagus degenerated after lesions of this nerve, and concluded that this posterior nucleus must be motor. Van Gehuchten has sought to explain this degeneration in another way. Dr. Spiller said that Dr. Dercum and he had just reported to the association a case of amyotrophic lateral sclerosis, in which the posterior nucleus of the vagus was degenerated, and the anterior was apparently normal. They had found a number of similar cases in the literature. In this disease the motor system is chiefly affected, and it is remarkable that the posterior nucleus of the vagus should present such evident signs of degeneration if it is a sensory nucleus.

Dr. Onuf, in reply to a question by Dr. Booth, said that in their experiments the thoracic sympathetic had been removed in two cases, and in both instances the animals developed diabetes.

Dr. F. W. Langdon was inclined to believe that the paper of Drs. Onuf and Collins would prove of great clinical importance. In myelitis, for example, we are all acquainted with the variability of the symptoms, and the speaker said that in locating such lesions he had always laid considerable stress upon the presence or absence of trophic symptoms. He had come to look upon the occurrence of marked trophic disturbances, bed-sores and similar conditions, as an indication of a lesion far back in the gray matter, and the investigations of Drs. Onuf and Collins give us a very satisfactory reason for this clinical fact.

Dr. Joseph Collins did not think it necessary to speak further of the experimental conclusions contained in the paper, but added a few remarks on the clinical aspect of the subject. In their experiments they had had in mind that if the sympathetic could be located in the spinal cord, certain symptoms of syringomyelia and tabes, about which we are now in the dark, could be easily explained. Not long ago he saw a boy, 13 years old, whose symptoms were diarrhoea, paroxysmal in character, which had extended over a period of several years; a condition of the right eye commonly known as the "Schultze eye," atrophy of the thenar and hypothenar eminences of the right hand, and cervicodorsal kyphosis. No sensory symptoms were noted. Dr. Collins said he hazarded the diagnosis of syringomyelia, despite the absence of sensory phenomena, and explained the symptoms in this case by the presence of a lesion in the central canal, which, in extending, implicated the paracentral nuclei and the nuclei of the intermediate zone, without encroaching upon any of the sensory fibres. The allo-

cation of the sympathetic to the medulla oblongata, which their results showed, threw much light on the interpretation of symptoms referable to the sympathetic system, occurring with bulbar disease and asthenic bulbar paralysis.

Dr. Onuf, in closing, said that several investigators have shown that all the dilating nerve fibres of the pupil are not derived from the cervical sympathetic; compensatory fibres being derived from the cranial nerves, probably the trigeminal.

He thought that the more we learn about the localization of the sympathetic nerve, the less shall we be inclined to diagnose syringomyelia in a diagrammatic or dogmatic way, and the more shall we be guided by a knowledge of the localization of the lesions. In syringomyelia it is not really the disease that makes the peculiar combination of the symptoms; it is the location of the process.

220. AMYOTROPHIC LATERAL SCLEROSIS. Raymond (La Presse Médicale, Nos. 41 and 43, 1897).

After a clinical demonstration of two cases (one male, one female) of this disease, which began with bulbar symptoms, and in which the arms and legs were later affected, the author discusses the relationship existing between amyotrophic lateral sclerosis, glosso-labial-laryngeal paralysis and progressive muscular atrophy of the Aran-Duchenne type. Certain authorities, headed by Leyden, hold that there is no spinal muscular atrophy depending upon a lesion strictly limited to the cells of the anterior horns, but that there is always more or less involvement of the fibres of the pyramidal tracts. Others of the school of Vulpian, of whom Dejerine is the chief representative, deny the existence of a glosso-labio-laryngeal paralysis due to lesion of the bulbar nuclei, without involvement of the pyramidal tracts in that region. Each of these views the author thinks incorrect. He cites an observation by Jean Charcot of a case presenting the typical symptoms of spinal muscular atrophy of the Aran-Duchenne type, in which lesion of the cells of the anterior horns, without involvement of the white columns, was found, and one of his own of a case of glosso-labio-laryngeal paralysis, in which the lesion was strictly limited to the nuclei of the bulb and concluded that while they may be closely related, the diseases in question must be considered as separate and distinct morbid entities. As to the lesion causing the rigidity and spasmodic phenomena in amyotrophic lateral sclerosis, the author expresses the opinion that it is not the sclerosis of the lateral tracts, but is probably a lesion in the gray matter of the cerebrum. As negative evidence, he mentions a case of Senator's, in which, though the clinical picture of amyotrophic lateral sclerosis, with involvement of the bulbar nuclei, was present, the atrophy disclosed degeneration of the bulbar and spinal nuclei, with diffused lesions throughout the cord, but no sclerosis of the lateral tracts. Positive evidence supporting his view he does not give.

ALLEN.

A SUMMARY OF THE SYMPTOMS IN SIXTY-ONE CASES OF LOCOMOTOR ATAXIA, WITH ADDITIONAL REMARKS,¹

By W. H. RILEY, M.D.,

Superintendent of the Colorado Sanitarium, Boulder, Col.

The sixty-one cases of locomotor ataxia, the most important symptoms of which are here given in the order of their relative frequency, have been examined and treated by the writer in sanitariums with which he has been connected. These patients were all males, which is quite uncommon for so large a number of cases. The percentage of females suffering with this disease is, however, usually small.

A history of syphilis was given in thirty-one cases out of forty-nine. Of the remaining eighteen cases of the forty-nine, fourteen had had gonorrhœa, been excessive in sexual indulgence, or gave other evidence of possible exposure to syphilis. Of the remaining twelve cases of the sixty-one, syphilis was either denied, or this point was not determined in the history of the case. In the cases that gave a history of syphilis, from two years to thirty years intervened between the primary venereal disease and the initial symptoms of locomotor ataxia. In most cases the initial symptoms of ataxia appeared from eight years to fifteen years after syphilis had developed. In two cases the disease followed soon after a mechanical injury. A history of exposure to wet and cold was given in seven cases. One case developed immediately after an attack of typhoid fever. This last case, however, differed from cases usually seen, in that the disease was not progressive in character, and the only prominent symptoms

¹Read at the twenty-fourth annual meeting of the American Neurological Association, May, 1898.

present were well marked ataxia in all four extremities, as well as in the muscles of speech.

The majority of these cases came from the middle and higher walks of life. In twenty-nine cases, the disease first showed itself between the ages of thirty and forty years; in twenty cases, between the ages of forty and fifty years; in six cases, between the ages of fifty and sixty years. In two cases the disease began at the age of twenty-five, and in one case, following typhoid fever, it began at the age of twenty-two years. The initial symptom, as given by patients in the history of their cases, was as follows:

In thirty-seven cases the initial symptom was pain in some part of the body, and was usually described as "rheumatic"; in three cases, it was gastric crises; in three cases, laryngeal crises; in four cases, incoördination of the lower limbs. In the remaining cases the initial symptom consisted of one or more of the following: Various paræsthesias in the extremities, diplopia, partial blindness, vertigo, difficulty in emptying the bladder, loss of sexual function, general feeling of weakness—frequently without exertion—and digestive disturbances.

The symptoms of the sixty-one cases tabulated in the order of their frequency are as follows:

1. Knee-jerk absent in.....55 cases.
2. Severe paroxysms of pain in.....54 cases.

In twenty-one of these cases severe pains were in the arm, trunk and legs; in fifteen, in the legs alone; in thirteen, in the trunk and legs; in one, in the arms alone; and in one, in the trunk alone.

3. Ataxia in locomotion, with eyes closed (56 cases examined), present in.....54 cases.
4. Various paræsthesias, as numbness, pricking, formication, etc., present in.....53 cases.

In thirty of these cases, paræsthesia was confined to the lower extremities; in twenty-one, to both lower and upper extremities; in two, there was paræsthesia in the face, as well as in the lower and upper extremities.

5. Static ataxia, with eyes closed (56 cases examined), present in.....54 cases.
 6. Anæsthesia (referring to tactile, temperature and pain sense, one or more being affected), present in.....45 cases.
- In fourteen of these, the anæsthesia was in the upper and lower extremities; in two, in the trunk and upper and lower extremities; in two, in the face and upper and lower extremities; in the remainder, in the lower extremities.
7. Girdle sensation about trunk present in....39 cases.
 8. Constipation present in.....39 cases.
 9. Static ataxia, with eyes open (56 cases examined), present in.....41 cases.
 10. Ataxia in locomotion, eyes open (56 cases examined), present in.....41 cases.
 11. Cold extremities in.....34 cases.
 - Loss of sexual power, partial or complete, in.35 cases.
 - General feeling of weariness, with diminished motor power, in.....37 cases.
 12. Area of hyperæsthesia about abdomen and lower part of trunk, with increased skin reflexes in the same area, in.....29 cases.
 13. Indigestion and stomach disorders in.....33 cases.
 - Considerable loss of weight in.....33 cases.
 14. Myosis present in.....28 cases.
 - Argyll-Robertson pupil (that is, pupil contracted, responding to light, but not to accommodation), present in.....28 cases.
 15. Nervous irritability and despondency in....25 cases.
 16. Accelerated pulse (85 or above) in.....25 cases.
 17. Skin reflexes (plantar, cremasteric and abdominal) increased in.....24 cases.
 - Incomplete retention of urine in.....27 cases.
 18. Insomnia in.....20 cases.
 19. Ataxia in arms in.....15 cases.
 20. Diplopia usually present at beginning of disease in.....14 cases.
 - Skin reflexes absent in.....16 cases.
 21. Delayed sensations in.....14 cases.
 - Partial incontinence of urine in.....15 cases.
 22. Ptosis (transient or permanent) in.....13 cases.
 - Attacks of sudden vertigo in.....13 cases.
 - Partial iridoplegia in.....11 cases.

23. Partial deafness in.....13 cases.
24. Plantar skin reflexes absent, or very much diminished, with other skin reflexes normally present, in.....10 cases.
- Gastric crises in.....11 cases.
25. Tenderness along the spine in.....7 cases.
- Diminished faradic irritability of muscles in..6 cases.
- Diminished galvanic irritability in.....6 cases.
- Unable to walk with crutches or two canes in6 cases.
26. Rise of temperature during paroxysms of pain in.....6 cases.
- Optic nerve atrophy in.....7 cases.
- Laryngeal crises in.....6 cases.
- Diarrhœa in.....8 cases.
- Pupils dilated in.....8 cases.
27. Unequal pupils in.....6 cases.
28. Polyæsthesia in.....4 cases.
29. Complete retention of urine in.....3 cases.
- Complicated with marked muscular atrophy with the electrical reaction of degeneration in muscles in.....2 cases.
- Tremor in hands in.....2 cases.
- Anæsthesia in face in.....2 cases.
- Taste affected in.....2 cases.
- Area of hyperidrosis about trunk in.....2 cases.
- Knee-jerk present and normal in.....2 cases.
- Knee-jerk present, but unequal, right stronger, in.....1 case.
30. Knee-jerk exaggerated in.....1 case.
31. Hyperidrosis of both legs in.....1 case.
- Knee-jerk present, but diminished, in.....1 case.
- Knee-jerk present in one limb and absent in the other.....1 case.
- Complicated with general paresis in.....1 case.
- Traces of albumin in urine in.....1 case.
- Complete incontinence of urine in.....1 case.
- Traces of sugar in urine in.....1 case.

With reference to the symptomatology of tabes, I believe that this is fairly well represented in a report of the symptoms found in sixty-one given herewith. Many of these are seldom noticed, or only lightly touched upon, by the majority of writers on this subject; and they are even

more frequently overlooked in examinations of patients suffering with this disease.

The symptoms apt to be overlooked in connection with this disease are an exaggeration of the skin reflexes, partial deafness, and accelerated pulse. In addition to these, there is in many cases insomnia, considerable loss of weight, with more or less disturbance of the digestive organs, and very frequently a relaxed condition of the abdominal muscles, allowing the stomach and bowels, and sometimes other abdominal organs, to become prolapsed and displaced. This is often a very marked condition in locomotor ataxia.

Another fact that may be noticed in this connection is that the knee-jerk is not always absent in locomotor ataxia, as is usually supposed. Of these sixty-one cases reported, it was found absent in fifty-five. Of the six remaining cases, it was present, and apparently normal, in two; present but unequal, the right being the stronger, in one; present, but much diminished, in one; present in one limb and absent in the other, in one case; and in one case it was certainly exaggerated. There is another class of cases of so-called ataxic paraplegia, in which the knee-jerk is exaggerated; but in this class there is an actual paralysis present in the lower limbs, in addition to the symptoms of locomotor ataxia, and the lesion in the cord occupies a larger and entirely different territory than in typical locomotor ataxia. The case here referred to, in which the knee-jerk was exaggerated, does not belong to this class. In this particular instance there was no paralysis of the lower limbs, and a careful study of the symptoms of the case would in no way warrant putting it in this class of so-called ataxic paraplegias.

Dr. S. Weir Mitchell, of Philadelphia, has reported a case in which the knee-jerk was exaggerated, but the symptoms of ataxia were in the upper extremities entirely.

Where the anæsthesia affects the arms and hands, I

have found in several cases that it was confined to the ulnar nerve. This observation has been made by others.

With reference to the exaggeration of the skin reflexes, in my own examinations I have found this condition in many cases which I have had an opportunity of examining at an early stage of the disease. As the malady progresses, the skin reflexes diminish, and finally disappear entirely, so that in most cases of tabes in the second or third stage of the disease the skin reflexes are entirely absent. I have found this exaggeration of the skin reflexes of great diagnostic value. Cases are frequently seen where, in the early stages of the disease, it is somewhat difficult to make an accurate diagnosis. The symptoms are not prominent, and, perhaps, a diagnosis must be formed upon only a general history of the case, together with two or three symptoms. In instances of this kind, when I have found the skin reflexes exaggerated, in connection with a few other symptoms pointing to this disease, I have usually had no trouble in making a diagnosis of locomotor ataxia.

I remember distinctly one case coming under my observation, where the only symptom pointing to incipient locomotor ataxia, with only the very slightest suggestion of incoördination, was decided skin reflexes, particularly plantar. The knee-jerk was present, and all other symptoms of the disease were absent. The diagnosis of incipient locomotor ataxia was made. I had an opportunity to watch this case for about two years, and, as time progressed, nearly all the other symptoms of locomotor ataxia made their appearance. I do not believe that in the literature treating upon this subject sufficient importance has been given to this symptom in the earlier stages of the disease.

The deafness which frequently occurs in those suffering with this disease I believe to be a part of the disease proper, and that it should be considered as forming part of the symptom-complex which characterizes the trouble.

The cause of the deafness is due to a lesion of the auditory nerve in some part of its course, similar to that which affects the roots of the spinal nerves. If these cases in which deafness is present are carefully examined, and the cause of the deafness sought, it will be nearly always found to be of nervous origin; at least, such has been my experience.

An accelerated pulse is another symptom which is usually overlooked. I have reported it present in 25 of the 61 cases here reported. In these 25 cases the pulse was 85 per minute or above. This is probably due to an irritation of the pneumogastric nerve, similar to that which in the spinal nerves produces sensory symptoms.

Another unusual symptom noted in connection with the study of these sixty-one cases was, in eight cases, a dilatation of the pupil, instead of a contraction, as is usually the case. In those cases in which the pupils were dilated, they did not respond to the light, and only very sluggishly to accommodation. In one of the sixty-one cases there was a trace of sugar present in the urine, which has been observed a few times in other cases of this disease.

These cases were seen in all stages of the progress of the disease, some in the first, some in the second, and some in the third stage. As the symptoms change as the disease progresses, the comparison of the symptoms found in any number of cases which may be made by one observer cannot well be compared with an equal number of cases of this disease made by another observer, for the reason that one observer may see more cases in a certain stage of the disease than another; and, secondly, the relative frequency of any symptom or group of symptoms may vary.

The relative importance of the different factors which are supposed to be the cause of the disease is a question with reference to which there is considerable difference of opinion. As nearly as can be determined by clinical observation, the factors which enter into the causation of

this disease may be briefly stated as follows: Neuropathic tendencies, syphilis, sexual excesses, exposure to wet and cold, overexertion, mechanical injuries.

Between the opinions of Erb, Möbius, Strümpell, Fournier and others, on the one hand, who regard tabes, in the majority of cases, at least, as purely a post-syphilitic condition, and those of Leyden, Charcot, etc., on the other hand, who disregard syphilis entirely, or give it but a small place in the etiology of tabes, there are many careful observers who hold all grades of opinion between these extremes. Leyden claims that syphilis must be left out of the etiology entirely; while Charcot strongly favored a neurotic heredity, and regarded syphilis of minor etiological importance. The doctrine of the syphilitic origin of tabes is mainly due to Fournier, of Paris (in 1876), and Erb, of Heidelberg (1879). Previous to either of these dates, however, Virchow, Wunderlich and Berger had already expressed themselves as believing that many cases of tabes were of syphilitic origin. In 1883, Erb declared that syphilis was the most important factor in the causation of tabes dorsalis, and that persons not previously infected with syphilis had but the slightest chance of suffering from this disease.

In the third number of his *Neurologische Beiträge* C. J. Möbius called attention to the changes of opinion of various authors since 1880 in regard to the etiology of tabes. He claims that the number of authors who regard syphilis as an etiological factor is constantly increasing. While Erb, Strümpell and other strong defenders of the syphilitic theory admit that a certain percentage of tabetics can with certainty be excluded, as not having had previous syphilitic infection, Möbius holds the most extreme views, and considers such an infection as the *sine qua non* for the development of this disease, and believes that Edinger overestimates the importance of excessive function as an etiological factor.

In 1892, Erb found that 89 per cent. of the cases of

tabes that he had examined, which included a large number, had previously had syphilis. Fournier states that over 90 per cent. of persons suffering from tabes have had syphilis. Out of 247 cases examined by Hirt, 90 per cent. were syphilitics. Senator gives 70 per cent., Mendel 75 per cent. Gowers, of London, found in his private practice that 58 per cent. of his tabetic patients had previously had syphilis, and he thinks that, could the facts be obtained, two-thirds would be nearer the truth. In Fraenkel's experience, the percentage was 50 7-10; in Gerhardt's, 50; in Dana's, 50. In a minute study of 212 cases, Lagondaky found 42 per cent. syphilitics, Eulenberg gives 36 4-5 per cent. In sixty-eight cases examined by Borgherini, he found that 32 per cent. had previously had syphilis, and 37 per cent. more had suffered from chancroid. A neuropathic taint was found in half his cases. De Werker gives 30 per cent. as syphilitics, while Panas finds that one-third of his cases have previously had syphilis.

Minor shows from his statistics that tabes is much more rare in Russia among the Jews than among other Russians, which is undoubtedly due to the fact that the latter are more or less syphilitic. Magel found 46 per cent. of syphilitics in 1,403 cases. In the experience of Westphal, 14 per cent. of his tabetic patients were syphilitic. In my own experience I have found that 60 per cent. of the cases suffering from tabes dorsalis had previously had syphilis.

Grimm, in the Berlin Medical Gesell., April 11th, 1894, gives the result of his seven years' experience in Japan, syphilis being very prevalent in that country. Of the 13,000 cases which came to his hospital, 1,020 were affected with syphilis. His expectation to find syphilitic tabes was not realized. He observed only five undoubted cases of tabes in Japan, of which only one had a history of syphilis.

Recently, Edinger has advanced a new theory as to

the origin of *tabes dorsalis*, as well as some other diseases of the nervous system. Edinger's theory is based upon the following laws:

1. The healthy normal activity of every organ always causes, or is accompanied by, certain molecular changes which take place in the organ. The organ consequently suffers certain damages, which must be repaired. When the restitution in the organ is equal to the amount of function, the organ is strengthened by functioning; but when not, it undergoes retrogressive changes.

2. A diseased, damaged or weakened tissue soon decays, and gives place to the surrounding healthy tissue, which grows into it and takes its place.

Weigert has proved that so-called hypertrophy, proliferation, interstitial inflammation, etc., are no more than the ingrowth of healthy tissue into the diseased tissue. In the so-called gray atrophies of the central nervous system the nerve tissue is always first diseased, after which it retrogrades, and is finally destroyed by the ingrowth of neuroglia. A degeneration of tissue must follow whenever there is a disproportion between function and restitution to such a degree that the tissue does not return to its former condition. Edinger thinks that, by properly appreciating these facts, many problems may be explained which heretofore have not been fully comprehended. He therefore holds that excessive function is a common cause of *tabes*, as well as of other diseases of the central nervous system.

Tabes is most frequently observed in persons who overexert their legs, such as military officers, railroad men, etc.; while it is seldom found in women of sedentary habits. In these cases the neuromuscular mechanism necessary for the maintenance of the equilibrium of the body and the normal gait is excessively active, and under an uncommonly high pressure. This also explains the rare occurrence of the disease in prostitutes, notwithstanding they are frequently affected with syphilis, the etiological im-

portance of which Edinger does not seem to underestimate.

The existence of tabetic symptoms in diabetes shows the intimate relation between diseases of the spinal cord and nutrition.

Mechanical injuries are usually considered by most writers on this subject as one of the causes of *tabes dorsalis*. The cases in which the disease has followed a mechanical injury, or in which there seemed to be a relation between an injury and the development of the disease, are comparatively few, and the evidence that the mechanical injury caused the disease, or was even a factor in the causation, is by no means conclusive.

Hitzig has recently analyzed sixty-six cases in which the cause of the disease was said to be traumatism. In these sixty-six cases, only ten or eleven met the requirements of sound criticism. Hitzig concludes from his study of these cases that the occasional onset of the disease in the injured side, or the preponderance of the symptoms of that side, are not sufficiently constant to warrant a conclusion of the traumatic origin of the disease; but, notwithstanding the great importance of previous venereal infection in most cases of *tabes*, Hitzig thinks that there are cases which show a distinct relation between traumatism and *tabes dorsalis*, and which cannot be explained in any other way.

In the February number of the *Journal of Nervous and Mental Disease*, 1895, Dr. Morton Prince gives a very interesting and valuable article on "Traumatism as a Cause of Locomotor Ataxia." In this article Prince describes quite fully two illustrative cases which show the liability to error in arriving at a conclusion in this matter, when the statements of the patient are taken as the basis for an opinion. Dr. Prince thinks—and the point is well illustrated in the two cases he reports—that a person may have locomotor ataxia for some months, or even several years, without knowing it, or knowing that anything is

specially wrong with him. Especially is this apt to be the case when the sensory symptoms are in abeyance. In view of this fact, Prince insists that the following rules must be rigidly adhered to in examining the evidence afforded by alleged individual cases:

"1. The subject must have been proved free from tabes, either immediately before or immediately after an accident.

"2. The subject must be shown not to have been exposed to other known causes, as syphilis, for example.

"3. The traumatism must have been of a nature to produce a physical or psychical impression of an appreciable degree, and not such a one as people are frequently exposed to without suffering afterward from tabes, e. g., the extraction of a tooth or a mild bruise.

"4. The symptoms must have made their appearance within a reasonable time after the accident—at least within a year.

"5. The diagnosis must have been established beyond a reasonable doubt."

In this article Prince analyzes critically the evidence which is given in cases thus far reported, in which traumatism was the supposed cause. He divides these cases into three classes.

The first class, consisting of twenty-two cases, he considers inadmissible on account of the triviality of the injury, the pre-existence of syphilis, long interval between injury and onset of symptoms, doubtful diagnoses, etc.

The second class of cases, consisting of six, are those which cannot be excluded, but which, from various circumstances, are questionable in evidence.

The third class are those in which the disease was apparently caused by traumatism. Of these, there are twelve cases. From those studied, Prince gives the following as the result of his investigation of this subject:

"Taking all the facts above collated into consideration, it would seem that the current view that locomotor ataxia

may be caused by traumatism, per se, irrespective of direct lesion of the cord, is not sustained by the evidence thus far adduced. If such a relation exists, further evidence is required before it can be accepted. It would seem to be more probable, aside from mere coincidence, that when a sclerosis of the posterior column develops after a traumatism, the subject was already doomed to this condition, the process having already begun, and that the traumatism, at most, but accelerated the symptoms, and, possibly, the anatomical process."

In the writer's opinion, there are two things, or conditions, necessary to the development of this disease in any individual. One of these is an organic predisposition to the disease; that is, a neuropathic condition—a low resistance in the nerve elements. The other essential condition is the presence in the blood and tissues of a toxin, which, in a very large proportion of cases, is of syphilitic origin. This toxin, in the case of syphilis, is constantly formed in the body after it once becomes infected, and keeps up a continuous chronic intoxication for a number of years, until finally the vitality of the tissues is overcome, and the symptoms of *tabes* begin to appear.

Other infections, both acute and chronic, have been known to cause this disease. Cases are on record in which *tabes* developed after infection from tubercular disease and also from leprosy. These infections, like that of syphilis, are chronic; that is, the germs of the disease remain in the system for a long period of time, and, as the result of the life and activity of these germs, ptomaines are constantly formed, and consequently the system is kept in a state of intoxication for a period of months, or even years. The disease may also follow or accompany infections of acute diseases, such as diphtheria, typhus and typhoid fever. It is a well known fact that in many cases of diphtheria there is a post-diphtheritic paralysis. This is quite frequently supposed to be due to a neuritis which is the result of the action of diphtheria toxins upon the nerve

fibres. This same poison may, and does, in some cases at least, produce tabes. In my own experience I have seen one well developed case of locomotor ataxia (tabes dorsalis) follow an attack of typhoid fever.

There is, however, an essential difference between cases of tabes resulting from chronic infection like syphilis or tuberculosis, and those of an acute infection like diphtheria, typhoid fever, etc. In the first instance, the disease, in the large number of cases at least, is progressive, which is a natural result if the body is kept constantly intoxicated by poisons that are formed by the infection of syphilis or tuberculosis. In the second instance, that is, where the disease develops from an acute infection, it becomes, after a period at least, regressive; it does not continue to progress as in the first instance. The best reason I can give for this difference is that in these cases poisons are formed in the body only for a limited period of time. The germs of diphtheria and typhoid fever remain in the system but a few weeks, and consequently the poisoning continues only for a limited space of time.

The question will naturally arise in the minds of some, whether these cases of so-called tabes dorsalis following acute infections, such as typhoid fever and diphtheria, are really true cases of tabes dorsalis, or whether the ataxic symptoms are due to a multiple neuritis, which frequently follows these infectious diseases. To this point I have but to answer that cases of tabes dorsalis have been reported by those of experience and careful observation. In my own personal experience I have seen only two cases of this kind, which appeared to me to be cases of tabes dorsalis, and not multiple neuritis.

It is as unreasonable as it is unscientific, in the light of modern pathology, to suppose that a man in the prime of life, thirty years of age, for instance, should be attacked with a disease like locomotor ataxia, which constantly progresses and becomes more severe in character and wider in extent, causing the individual to stagger through

life, and finally totter into the grave, except there be in the body a cause that is constantly present and active. Certainly no good reason can be given why in a man in apparently good health there should begin to be formed on the posterior columns of the spinal cord a strip of sclerosed tissue, which constantly extends its borders, unless, as said before, there exists in the body a cause that is constantly acting. To say that this might be caused by perverted action on the part of the tissues—a sort of habit, as it were—is a very poor and unsatisfactory explanation. It does not give any reason why the disease should constantly progress as it does.

Besides the infections above referred to, chemical poisons may cause this disease. It is well known that poisoning from ergot, also from lead and mercury, is frequently followed by symptoms of *tabes dorsalis*. Besides these, poisoning from substances formed within the body in gouty, rheumatic and diabetic diatheses is sometimes followed by symptoms of locomotor ataxia. Locomotor ataxia may also follow pernicious anæmia.

I do not believe that mechanical injury, exposure to wet or cold, overwork, or sexual excesses without infection can be regarded as an exciting cause of *tabes dorsalis*, per se. None of these influences, acting alone or in conjunction, could cause a bundle of nerve fibres to degenerate, and the adjacent connective tissue to increase until a well defined, triangular band of sclerosed tissue is formed for a greater or less distance along the posterior part of the spinal cord. That these agents have some influence in exciting the disease in many cases is granted, but their action is in lessening the vitality of the nerve elements, and preparing them for another more active agent, which I believe is always a poison from some of the sources previously mentioned. The essential thing is a toxin in the blood, which may be a ptomaine, a leucomaine, or a chemical poison, either organic or inorganic.

Much valuable work has been done recently with ref-

erence to the pathogenesis of tabes. The old theory, which considered the primary lesion in tabes as a degeneration of the posterior columns of the spinal cord, will, in the light of recent research, hardly hold. That there is a degeneration of the nerve fibres of the posterior columns of the cord is a well established fact; but is this primary? Or is it the result of another lesion, which, at least in a casual relation, antedates it?

Rindfleisch ascribes the origin of tabes to a slightly progressive inflammation of the pia mater, which by reason of tissue continuity gradually extends to the connective tissue of the cord, and there forms an interstitial myelitis, which results in a secondary degeneration of the nerve fibre of the cord. Obersteiner and Redlich have recently advocated a modification of this idea. They have attempted to determine the exact starting point of the degeneration. By making oblique sections of the spinal roots in the direction in which they enter the cord, they have found that, normally, the roots are constricted by a circular band of connective tissue of the pia mater.

In the early stages of tabes, the roots of the spinal side of the restriction are found to be degenerated; the cause of this degeneration is a hyperplasia of the connective tissue, which forms the constriction. This presses on the nerve fibre, and leads to its degeneration. The reason that the nerve fibres in Lissauer's boundary zone of the spinal cord differ so in the disease is that in the spinal roots these fibres are located in the periphery, and consequently suffer most from the constriction. Nageotte, in the *Société de Biologie*, November 10th, 1894, gives his views of the primitive lesion of tabes. He believes that the initial lesion of tabes is a perineuritis, which affects the posterior spinal roots between the spinal ganglia and the point where the roots enter the subarachnoid space. The perineuritis at first partakes of the nature of an embryonic process, but later is fibrous in character. Following the inflammatory process is a degeneration of the root fibres,

which extends into the posterior columns of the cord. The anterior as well as the posterior spinal nerve roots are affected by the inflammation; but, as they seem to have greater resistance, the motor nerve fibres are less liable to degenerate.

Prof. Leyden, as early as 1863, expressed the opinion that the lesion in tabes begins in the posterior roots, and that the degeneration in the posterior columns of the cord is secondary. The researches of Obersteiner, Redlich and Nageotte, previously referred to, as well as those of the French neurologists, Marie and Dejerine, concur in the opinion expressed so long ago by Leyden. As to the primary lesion in tabes, Marie, however, holds that it is in the ganglia of the roots, and not in the root fibres.

It seems quite well established, therefore, that the disease begins in the posterior spinal roots, either in the nerve fibre or the ganglia, the nature of the lesion being at first inflammatory, and affecting the connective tissue, and, later, degenerative, affecting the nerve fibres.

The nerve fibres which form the posterior column of the spinal cord are but the extension of those which make up the posterior spinal roots.

A lesion affecting the fibres of the posterior spinal roots would likewise affect those of the posterior columns of the cord. A lesion of the posterior roots would interfere with the nutrition of the fibres of the posterior columns of the cord by cutting them off from their centre of nutrition, which is the spinal ganglia of the posterior roots.

With reference to the treatment of this disease, space will not allow a minute description. I can make only a few general suggestions. My own practice for some time past has been to direct the treatment along two lines, viz., (1) to eliminate poisons that are in all probability being constantly formed; (2) to improve the general health and nutrition of the patient, special treatment being directed to the seat of the lesion.

The first of these lines of treatment, that is, the elim-

ination of the poisons from the body, I believe can be best accomplished by the free use of water internally. I usually instruct my patients to drink from five to seven pints of water daily, and to take it in small quantities and at short intervals between meals. This flushes the tissues, and keeps the poisons that may be in the body in solution, thus favoring their elimination. It also increases the activity of the eliminative organs. In many cases it is best to have the patient drink the water hot. Besides this, the use of warm baths, particularly the electric light bath, which is usually very agreeable to these patients, is to be highly recommended. This does good, not only by the elimination of poisons, but also by relieving the distressing pains, which are so troublesome in this disease. By correcting all bad habits, prohibiting the use of tobacco and alcohol, and placing the patient on an aseptic diet, the further intoxication of the body from without becomes almost impossible.

The second indications for treatment are best met by the proper use of hydrotherapy, electricity in its various forms, massage and other manual and mechanical movements, including suspension treatment. I am satisfied from the results of the treatment in a large number of cases of this kind that a great deal more can be done for patients suffering with this disease than is usually supposed. I do not myself lay claim to any superior wisdom or extraordinary skill in managing these cases; but I believe from my own experience that when they can be put under proper conditions—the daily life of the patient controlled, and the treatment above indicated intelligently applied—as much can be accomplished in this malady as in many other diseases affecting other parts, which are generally considered less intractable.

It is certainly unjust to tell a man suffering from locomotor ataxia, in the first or second stage of the disease, that nothing can be done for him; I have had many cases concerning which such statements had been made by phy-

sicians, and yet, after taking the course of treatment as indicated, they have materially improved, the disease being checked in its onward progress, and the patient put in a position where he could be of practical service to himself and to his friends. And this is as much as, or more than, is done in the treatment of numerous other diseases which are not supposed to be as formidable as *tabes dorsalis*.

221. EIN BEITRAG ZUR PATHOLOGIE UND PATHOLOGISCHEN ANATOMIE DER TRAUMATISCHEN RÜCKENMARKSERKRANKUNGEN (SOGENANNTHE HÄMATOMYELIE, SECUNDÄRE HÖHLENBILDUNG. (A Contribution to the Pathology and Pathological Anatomy of the Traumatic Diseases of the Spinal Cord, etc.) Lax and Müller (*Deutsche Zeitschrift für Nervenheilkunde*, 12, 3 and 4).

A man became paralyzed in all his limbs immediately after a fall. Complete anæsthesia reached as high as the axillæ. The movements of the head and neck were not interfered with. Cerebral or bulbar symptoms were not noted, and consciousness was preserved even immediately after the accident. Motion in the shoulders and elbows gradually became possible, but the hands remained much paralyzed. The complete anæsthesia gradually changed to analgesia and thermo-anæsthesia. Tactile sensation was regained in the entire body. A gradually developing atrophy of the interosseous muscles of the hands and of the extensors of the forearms was noted. The muscles of the lower extremities became rigid, and all the tendon reflexes were exaggerated. Death occurred three years after the fall.

The vertebral column was intact. A cavity was found in the posterior part of the cord, and was limited to the fifth cervical segment. The lateral columns were sclerosed. Bands of tissue, consisting of glia fibres, vessels and medullated nerve fibres, were found within the cavity, and bore much resemblance to peripheral nerves. Secondary ascending and descending degeneration was noted. No remains of a former hemorrhage could be observed, but the cavity was believed to be due to the absorption of a hemorrhage and disorganized nerve tissue. A recent case of hæmatomyelia resulting from fracture of the vertebræ is also noted. The writers believe that the lower cervical region is a frequent seat of spinal hemorrhage. They think that a force which is sufficient to cause hemorrhage causes injury also of the nervous tissue, not depending directly on the hemorrhage. The case is important, as showing the development of syringomyelia after trauma.

SPILLER.

ON REGENERATION OF NERVE FIBRES IN THE CENTRAL NERVOUS SYSTEM,¹

By W. L. WORCESTER, M.D.

Dr. Worcester read a paper on this subject and exhibited photographs and specimens. The literature contained but few observations countenancing the belief that such regeneration takes place. Brown-Séquard and Eichhorst and Naunyn had reported regeneration with partial restoration of function in experimental sections. Stroebe had reported growth of nerve fibres from the posterior roots into the cicatrix, and also what appeared to be newly formed fibres in the anterior and lateral columns after division of the spinal cord in rabbits. Borst, in a case of fracture of the vertebral column, after three years found medullated fibres, which he compared to amputation neuromata, and which appeared to be outgrowths from the anterior and posterior nerve roots.

The case upon which the paper was founded was that of a woman, presenting paresis and partial anæsthesia of the left side, in whom, at the autopsy, the right corpora quadrigemina were found to be in a cicatricial condition, probably resulting from thrombosis. In the middle of the cicatrix a group of greatly contorted bundles of medullated fibres was found, entirely different from anything normally seen in that situation. The only connection with sound tissue that could be discovered was by a number of small bundles of fibres from the tegmentum, in the neighborhood of the red nucleus. The general appearance was very similar to that of an amputation neuroma, to which the author believed it to be analogous, in view of its dissimilarity to any normal structure, and the improbability

¹Read at the twenty-fourth annual meeting of the American Neurological Association, May, 1898.

of its being exempt from the destruction of the surrounding nervous substance in that situation.

DISCUSSION.

Dr. Joseph Collins said that, as he understood the condition described by Dr. Worcester, there was a bundle of nerve fibres in the degenerated area, which Dr. Worcester was not able to trace to any particular destination. He would like to ask if serial sections were made and, consequently, at different axial planes. If they had all been made on the same plane, and the author had been able to trace the fibres for a long distance, then the interpretation he had put upon the condition would be justifiable, but, if they had not been so made, a bundle of undegenerated fibres, coming from an origin cephalad to the lesion, and unimplicated by it, might have been encountered in certain sections cut in different planes from other sections.

Dr. Worcester, in reply to Dr. Collins, said the sections were all made at the same time, in an uninterrupted series, extending about three mm.; the abnormal group of fibres was largest in the centre, and grew gradually smaller at both ends. The speaker thought it absolutely certain that the condition found did not represent a normal tract of fibres. He had never seen anything like it in the normal corpora quadrigemina. It was either an abnormal growth existing previously to the lesion, or a new formation developed subsequently. On the former hypothesis, we must assume that this abnormal bundle of fibres possessed greater vitality than the surrounding normal tissues.

Periscope.

With the Assistance of the Following Collaborators:

CHAS. LEWIS ALLEN, M.D., Wash., D.C. R. K. MACALESTER, M.D., N.Y.
J. S. CHRISTISON, M.D., Chicago, Ill. J. K. MITCHELL, M.D., Phila., Pa.
A. FREEMAN, M.D., New York. H. PATRICK, M.D., Chicago, Ill.
S. E. JELLIFFE, M.D., New York. JOSEPH SAILER, M.D., Phila., Pa.
WM. C. KRAUSS, M.D., Buffalo, N.Y. HENRY L. SHIVELY, M.D., N.Y.
W. M. LESZYNSKY, M.D., New York. A. STERNE, M.D., Indianapolis.

ANATOMY AND PHYSIOLOGY.

222. CORTICAL LOCALIZATION IN ANIMALS. *British Medical Journal*, November 20th, 1897.

Wesley Mills, of Montreal, after a very full series of experiments, reaches important conclusions as to differences of cerebral localization in the lower animals, conclusions that are suggestive not only as to the evolution of cortical representation of definite movements, but as to localization in the human brain. For instance, he finds no defined centre for the hind limbs of the rabbit, and in this connection remarks that the method of locomotion in rabbits is peculiar, and not comparable to that of the rat, guinea pig, etc.

He comes to the following general conclusions:

In the dog, cat, rabbit, cavy, rat and mouse electrical stimulation of the cerebral cortex over definite regions produces regularly certain movements. These animals are, however, not on the same physiological plane with regard to this subject. The dog and the cat are more closely related, and fall into a physiological group by themselves. The rabbit, the cavy, the rat and the mouse constitute another group. There are well defined differences for the cat and the dog. The same applies to the members of the other group. In the cat and the dog the motor areas are better defined than in the members of the other group.

In the case of all these animals it has been clearly demonstrated that all motor centres are not functional equivalents—some respond more readily and produce better defined results than others. They seem to be better organized. There appear to be all degrees of this functional variation down to zero. The rabbit is an especially good illustration of some phases of this principle.

The cortical localization mapped out by Ferrier for the dog, cat, rabbit, cavy and the rat is in the main confirmed by the present investigator, but considerable allowance must be made for individual differences, and it is important, as has been just pointed out, to recognize that all motor centres in the same animal are not functionally equivalent in the sense explained above.

The removal of motor centres in the animals made the subject of this investigation does not lead to complete loss of the correspond-

ing movements, and in some cases the difference between the intact animal and that operated on is, after a few days, relatively slight; so that it is plain that motor centres in such animals are not strictly comparable with motor centres in the primates. In other words, here again the question of degree of localization and functional organization (among others) must be considered.

The bird is on a wholly different plane. None of the ordinarily recognized movements on stimulation of the cerebral cortex can be excited in the bird. On the other hand, certain eye movements, both intrinsic and extrinsic, follow as a result of stimulation of the cortex.

PATRICK.

223. RECHERCHES CLINIQUES SUR L'ALCALESCENCE DU SANG ET LES INJECTIONS DE SOLUTIONS ALKALINES CHEZ LES EPILEPTIQUES (Clinical Researches on the Alkalinity of the Blood, etc.). R. Charon et E. Briche (*Arch. de Neurologie*, 4, 1897, p. 465).

The author comes to the following conclusions as the results of a series of experimental researches:

1. In epileptics in the course of each quotidienne revolution the degree of alkalinity of the blood is modified regularly with minimum and maximum amounts corresponding with the digestive operations.
2. The convulsive attacks present nearly constant variations, isochronous and in inverse relationship to the variations of the alkalinity of the blood.
3. Repeated injections of alkaline solutions do not modify in any permanent measure the grade of alkalinity of the blood. It produces but a temporary rise in this alkalinity, which disappears within an hour, and during which no convulsive attacks are present.
4. Injections would seem to diminish isolated attacks and to augment those which appear in a series. The total number of attacks is not diminished.
5. The injections seem to augment the post-epileptic psychical manifestations, and in certain cases provoke delirious or maniacal attacks.

JELLIFFE.

224. RECHERCHES EXPERIMENTALES SUR LA THYROIDINE (Experimental Investigations on Thyroidine). Barteit (*Sitzungs. d. Naturf. Ges. z. Inojew*, 1896, p. 123).

The author, experimenting on himself and on animals with preparations of thyroidine, in powder form and alcoholic solution, reports:

I. Personal experiments. The ingestion of 0.006 gr. of pure thyroidine gives rise to a considerable diuresis, due in all probability to increased combustion of the carbohydrates, fatty substances, and albuminoids. There were no abnormal manifestations, nor increase in the pulse rate.

II. Experiments on animals. Even in large doses there was no evidence of toxic symptoms, and the diuretic action was but slight.

III. Investigations on isolated organs with artificial circulation. Thyroidine has no influence on the blood-pressure in cats, nor on the cardiac movements in frogs. In 0.006 gr. doses there was no action on the renal vessels, and consequently no increase of urine secretion in oxen.

Conclusions. The author considers thyroidine as the best tolerated, the least noxious, and the most constant preparation of the thyroid gland. It is not a real diuretic, its action upon the secretion of urine being indirect. It may be prescribed with benefit in obesity, in which metabolism is sluggish, and in all cases in which the function of the thyroid gland is impaired.

MACALESTER.

CLINICAL NEUROLOGY.

225. ON A CASE OF ACUTE MYELITIS. Dr. Jaccoud (Medical Week, September 17th, 1897).

Dr. Jaccoud discusses the diagnosis of hemorrhage into the cord in the report of a case in which the mistaken diagnosis was not refuted until the post-mortem examination. The patient suffered a sudden chill when perspiring freely, and eighteen or twenty-four hours afterward found that he could not pass water; no other symptoms had preceded this trouble. On the same day his legs began gradually to grow weak, until there was complete paraplegia, twenty-four hours after the retention began. On the third day an ulcer of small dimensions appeared on the sole of the right foot. Two other ulcers, one on the trochanter and one on the buttock, followed quickly, both on the right side. The rapidity of onset was the point upon which the diagnosis rested, and the immediate appearance of atrophic symptoms only added force to the probability of the diagnosis of hæmatomyelitis. The patient's history being absolutely good in respect to intoxication or infection of any kind rendered it probable that the original cause was the sudden chill, and that alone. He had no fever, and no symptoms other than those which were due to pressure in the lumbar portion of the spinal cord. There was no disturbance of sensation whatever, the skin reflexes were not affected, but the tendon reflexes were abolished. The preservation of sensation is rare in such cases, and would certainly have been very extraordinary had signs of hemorrhage been found in the cord. It was only to be explained by the supposition of Brown-Séquard that the posterior white tracts might convey sensation when the posterior gray substance had been destroyed. Dr. Jaccoud concluded the diseased regions were the white anterior and lateral columns and the anterior and posterior gray matter. A sudden ascending degeneration carried off the patient within a few hours by implication of the respiratory centre, and the somewhat unsatisfactory statement is made that the postmortem discovered a "focus of softening in the dorso-lumbar region of the spinal cord, measuring about seven centimetres in length." No trace of hemorrhage could be detected. Bacteriological examination showed the presence of streptococci and staphylococci, but no suggestion is made as to the manner in which the microbes gained entrance to the organism. It is a curious omission in the report of the case that more details should not be given as to the distribution of the focus of softening, as, in view of the ante-mortem statements of the regions probably diseased, comparison would be interesting.

MITCHELL.

THE
Journal
OF
Nervous and Mental Disease

Original Articles.

ON SCLERODERMA AND CHRONIC RHEUMATOID ARTHRITIS.¹

By F. X. DERCUM, M.D.,

Clinical Professor of Nervous Diseases in the Jefferson Medical College,
Philadelphia.

Our knowledge of some of the trophic diseases is as yet so obscure that new observations concerning them cannot but be of value. Several of them, especially acromegaly and myxœdema, have in the past few years received a large share of attention. Scleroderma, on the other hand, has not been studied to the extent that its importance deserves. This is also true of that other obscure disease, chronic rheumatoid arthritis. Repeated observations have convinced me that as regards scleroderma, our conception is as yet too limited. Our point of view has in the past undergone several important changes. The first was the recognition that all forms of local scleroderma and general or diffuse scleroderma are one and the same affection. The second consisted in the recognition of the fact that the disease process is not necessarily limited to the skin, but may include other structures as well. In a paper,²

¹Read at the twenty-fourth annual meeting of the American Neurological Association, May, 1898.

²Journal of Nervous and Mental Disease, July, 1896.

read some two years ago before the Neurological Section of the New York Academy of Medicine, I laid emphasis upon the fact that tendons, muscles, fasciæ, bones and joints may also become involved. That this involvement of tissues other than the skin may, in rare instances, be excessive, there can be no doubt. Indeed, the question arises whether they may not at times exceed those in the skin. The changes in the latter have so long been regarded as the primary and all-important features of the disease, that changes in other structures have not only been looked upon as secondary, but even as dependent upon the changes in the skin. Thus, most writers refer the restricted movements of joints or the atrophy of bone, as seen in the fingers in sclerodactyle, to the contracture of the overlying dermal structures and to the interference with nutrition thus produced. For instance, the impairment of movement in the fingers, of the wrist and of the elbows, appears in many cases to be directly dependent upon the contraction of the skin. In sclerodactyle, again, the fingers become for the most part thin and tapering, and when such cases are skiagraphed, we may find that the phalanges, especially the distal phalanges, have become decidedly pointed or sharpened, as though they were undergoing atrophy.³ In such cases the atrophy of the bone appears to be general, and it does not seem unreasonable to suppose that the changes in the bones are directly dependent upon the changes in the skin. The latter may be dense, hard and contracted, and may interfere greatly with the blood supply of adjacent structures. We should bear in mind, however, that changes may occur in phalanges when the skin is not hard and contracted, but merely thin and atrophic. This was true of a case studied by me several years ago, and which I have already reported.⁴ In this case the proximal and middle phalanges

³ Loc. cit. Addendum.

⁴ Loc. cit.

of the little finger of the left hand had almost entirely disappeared, while the skin was much wasted but not infiltrated, the finger being abnormally mobile because of the loss of bone. Many of the phalangeal joints also were ankylosed, the ankylosis being evidently due to fixation of the joints independently of the changes in the atrophic skin. Again, that changes occur in the bones of the fingers, independent of contracture and atrophy of the skin, is proven beyond all doubt by the following case.

Case I.—A. G.; female; married; aged 44 years; house-keeper.

Family history, negative; father, mother, several brothers and sisters all living and well; no history of nervous or skin affections.

Personal History.—Was perfectly well as a child and young girl; lived out as a servant; was compelled to do a great deal of washing, and frequently worked in the open air with her hands and face exposed, while wet, to intense cold; menstruated at fourteen; married at thirty years, but never became pregnant.

When twenty-six years of age, she noticed that the middle and ring fingers of the right hand were becoming stiff and swollen, and that this condition was more marked at times. A little later the affection made its appearance in the corresponding fingers of the opposite hand, and, finally, all the fingers and both thumbs became involved. Very soon the stiffness and swelling became decided, and the fingers became chronically swollen and thickened, so that it was impossible to flex or extend them in the normal manner. About this time also the face became hardened and stiffened. Distinct swelling does not appear to have been present. There appeared to be merely an infiltration, followed by a gradual shrinking of the skin. The lips became much thinner, while the skin of the cheeks and forehead was tense. Gradually, also, infiltration of the skin of the back of the feet, and slightly of the toes, became noticeable. This, however, has never been as marked as in the hands.

At various times small trophic ulcers made their appearance upon the first phalangeal joint of the little finger, and, after having existed for some time, slowly healed. Gradually the finger ends became more swollen than the rest of the finger, so that the latter became club-shaped. At the same time the tips of the fingers became shorter. Ulcerations

frequently occurred about the root of the nails. The nails became shortened and flattened. Upon one digit, the right forefinger, the nail was lost altogether. These ulcerations, the patient states, were always very painful.

Some time ago the teeth of the upper jaw gradually loosened, one by one, and had to be removed.

In other respects her health has been fairly good. She has suffered at times from mental depression, and quite frequently from headache. Vertigo and tinnitus were not at any time present.

Menstruation, which had been regular and normal up to



FIG. I. Sausage-shaped fingers in sclerodactyly with trophic changes in nails and distal phalanges.

two years ago, had ceased at that time, and had not since returned.

Status Præsens.—Patient presents the facies of scleroderma. The skin of the cheeks, forehead and lips is tensely drawn. The forehead presents an erythematous flush. Persistent pressure produces marked pitting over the forehead. No pitting can be elicited over the rest of the face. When the patient talks or smiles, it is readily seen that the mouth is much contracted; the lips become tense and thin. There is decided palor of the tongue, roof of the mouth and fauces.

There are no changes in the trunk or limbs other than

those described, save in the skin over both shoulders, which is somewhat infiltrated and hard. There is everywhere an absence or diminution of the superficial fat. In the hands there is slight infiltration of the dorsum and very marked infiltration of the fingers and thumbs. The fingers are more or less fixed in the semi-flexed position, and are thick, sausage-shaped or club-shaped. The distal phalanges are evidently shortened by atrophy. All of the fingers reveal traces of pre-



FIG. II. Skiagraph of the fingers of the right hand showing changes in the distal phalanges.

vious ulceration in the matrix of the nail, save the little fingers, the nails of which are apparently normal. On the forefinger of the right hand the nail has been entirely lost, a scar of the matrix only being left. An ulceration is at present active at the root of the nail of the left thumb. This ulcer is exceedingly painful. Tactile, thermal and pain senses are everywhere preserved. Knee-jerks are not changed. An examina-

tion of the blood failed to reveal any evidences of leucocytosis. Examination of the urine, negative.

Some two months after the first examination the patient again presented herself with an ulcer over the left olecranon, while extreme ulceration had recurred on the fingers. Ulcers were present on all of the fingers, the little fingers being this time also affected. The ulcers involved the matrix of the nails and also the tips of the fingers. They were all very painful.

Seen again four months later, the ulcers were evidently in process of healing. The little fingers had become much contracted, and the mouth was also more drawn. Backache was also complained of, and the general health had evidently become much impaired. The patient complained also of cold sensations and occasional flushes.



FIG. III. Skiagraph of the left thumb and forefinger.

In this case the changes in the face were so typical that there could be no doubt as to the nature of the affection. The face was drawn, the cheeks flattened, the angles of the mouth slightly drooping and the lips contracted. The hands were also in a condition of sclerodactyle. All of the digits were fixed and rigid, but instead of being pointed and showing excessive contracture and atrophy of the skin, they were, as is seen in the photograph (Fig. I.), enlarged, bulbous and sausage-shaped. Ulceration, as has already been described, had taken place in the tips of the digits and thumbs, with loss of the soft tissue and also with loss of some of the nails. When these fingers were skiagraphed (Figs. II. and III.), a most interesting condition of the bones was revealed. The changes were limited to the distal phalanges. There was not the general sharpening and wasting shown in the first case skiagraphed

by me⁵, but, instead, there had ensued a gross and very decided loss of bony tissue, and in several of the digits, for example the thumbs, in which the nails had been fairly well preserved, and in which there had been no wasting of the pulp of the tip, very striking changes were revealed in the bones. The changes were of such a character as to justify no other inference than that they were trophic in character. In this connection it is interesting to recall the fact that Wolters described, in a case of sclerodactyle, which he examined microscopically, an interstitial inflammation of the phalangeal bones. As is well known, symptoms suggesting Raynaud's disease are every now and then observed in sclerodactyle. In this case, however, no such symptoms were present, and there was no history suggesting vascular crises. The case is further interesting because the trophic changes in the fingers strongly call to mind those of Morvan's disease. From the latter affection, however, it is sharply defined by the absence of all sensory losses, all forms of cutaneous sensibility being preserved, and by the presence of pain in the ulcers.

The following case which must be placed under the caption of rheumatoid arthritis—whatever that may be—presents a number of features pointing strongly to scleroderma, and suggesting a similarity in the pathological changes at work. The case is specially interesting when we reflect that it is not improbable that under chronic rheumatoid arthritis, two or more clinical entities may be confused.

Case II.—E. McG., male; aged 28; born in this country; inmate of nervous wards, Philadelphia Hospital.

Family History.—Father died of pneumonia at 46; mother living and well; one brother and three sisters living and well; one brother and two sisters died in infancy; no history of rheumatic, skin or nervous affections in the family.

Previous History.—Was well during childhood, save that he frequently had attacks of croup; also had measles; frequently suffered from headache; had good health otherwise up to fifteen years. At that time had a swelling of the right knee, which confined him to bed for one week; the attack was not accompanied by pain. It was pronounced, according to patient's statement, to be "rheumatism and white swelling." One year later the swelling recurred, and he was confined to

⁵ Loc. cit. Addendum.

bed two weeks. One and a half years later the attack again recurred; this time it lasted three months, and was accompanied by pain. Seven months later, in June, 1887, he suffered from another attack. The right ankle began to swell, and became painful, so that he could not walk. This time the attack lasted three weeks, but subsequently he became, as he thought, entirely well. Some time later, however, he began to feel stiff all over; had occasional "catching" of the muscles of the left thigh, half way between hip and knee. He would be so stiff at times that he could not walk. His general strength also suffered severely; weakness became marked. In about a year—1888—he was able to use his legs but little on account of stiffness, though this stiffness varied considerably from day to day. Both feet also became somewhat swollen



FIG. IV. (Case 2.) Showing general position of limbs.

in the daytime, the swelling disappearing on going to bed. His arms, neck and back also began to get stiff about this time, and these symptoms gradually grew worse. He was treated at various times with massage, but passive movements gave him pain. For a time, in 1894, his hands were swollen, but this swelling subsequently disappeared. About this time, also, he suffered severely from neuralgic pains in the head. Little by little the stiffness increased, until his limbs, hands and feet became more or less fixed in position. About three years ago the skin of the legs became smooth and shiny, and subsequently the skin in other situations assumed a similar character. About this time, also, the jaw became quite stiff. He has not been able to feed himself for five years. He has been unable to walk or even stand up for eight years.

Status Præsens.—Patient lies extended upon his back in bed. Some diffuse muscular wasting, with loss of superficial fat, has evidently taken place. No local muscular atrophies are, however, apparent. Both legs are in a position of extension, the feet in extreme extension. The amount of voluntary movement in the legs is extremely limited. The patient is able, by the action of the thigh muscles, to move legs upward to an extremely limited degree—a mere fraction of an inch. On attempting to make passive movements with either leg, it is found that the ankle joint, tarsal and metatarsal and



FIG. V. Right hand in Case 2.

knee joints are absolutely fixed. There is also fixation of both hip joints, though here the fixation is not absolute. Very slight movement in antero-posterior direction, as just stated, can still be made. On attempting lateral movement, however, the limbs cannot be moved without moving the entire pelvis. The right knee joint is decidedly enlarged, the patella quite prominent. The circumference of the right knee joint is $12\frac{3}{4}$ inches, of the left knee joint $11\frac{1}{2}$ inches. The right leg is not so completely extended as the left; it is very slightly flexed at the knee. The right foot is fixed in extension; its toes are in a position of marked abduction and slight flexion. The right

tarsus is thickened. The left foot is also fixed in extension; its great toe is extremely flexed, but not abducted or adducted; its second toe is markedly adducted and crossed over the great toe; the remaining toes of the left foot are slightly adducted, and neither flexed nor extended.

The arms are extended, but not completely so. There is absolutely no movement at the elbows or at the wrists. There is very slight voluntary movement at the shoulders in all directions. Upon passive movement, it is found that the excursion of the arm is considerably greater than by voluntary movement, and also that there is more movement in the right shoulder than in the left. The movement does not, however, take place in the shoulder joint, or, at least, only to a very small extent, for, on attempting to ab-



FIG. VI. Left hand in Case 2.

duct or adduct the right arm, the scapula at once moves with the humerus; this is even more marked in the left shoulder. The patient is able to perform very feeble movements of flexion and extension in the fingers of the right hand. The fingers are much distorted. The proximal phalanges of the fingers of the right hand are in a position of overextension, while the middle and distal phalanges are in marked flexion; these peculiarities are most marked in the little and ring fingers; the middle and forefinger are also extended, but to a less degree. The middle phalanges are flexed at a right angle, while the distal phalanges are again in a position of extension. The fingers are spread

apart at their metacarpal articulations, the little finger being much abducted, while the forefinger is decidedly adducted, as are also the middle and distal phalanges of the fore and middle fingers. The thumb is extended, adducted and displaced, so as to occupy almost the same plane as the palm of the hand. Passive movements about equal the small voluntary movements described. In the left hand the fingers are likewise much distorted; the thumb is extended and adducted; the proximal phalanges of the ring, middle and fore finger are flexed, especially the latter; the middle and distal phalanges are also flexed in varying degrees; the middle finger is adducted so as to cross the tip of the thumb; the little finger is in the position of moderate flexion at the middle phalanx. The palmar surfaces of both hands are much indurated, and hard to the touch. Both wrists are thickened.

The head and the entire trunk are rigid in extension. The head can be slightly raised from the pillow; slight extension of the head backward is also possible. Movement of the entire head is, however, so limited that when the pillow is withdrawn the head remains fixed and extended upon the trunk. There are almost no lateral movements of head, and very slight rotary movement. The jaw is so firmly fixed that the teeth can only be separated for a quarter of an inch.

The trunk is so fixed that it is impossible to obtain any movement in any direction. The ribs are almost completely fixed in position; no movement is perceptible in any but the false ribs, and in these only upon forced respiration. The thorax everywhere feels firm and resistant to touch, the intercostal spaces offering almost as much resistance to touch as the ribs.

Respiration is almost exclusively abdominal. The abdominal muscles move freely only upon forced inspiration. They feel firm, hard and resistant to the touch, suggesting almost the board-like feel met with in tetanus. The abdominal reflexes are much exaggerated.

The muscles of the neck feel excessively firm and hard, with the exception of the right sterno-mastoid, which is soft and flaccid. The right deltoid, even in a flaccid condition, seems to have its density increased, and on efforts to move the arm it becomes excessively hard. The biceps and triceps are flaccid, yet likewise present a firm, fibrous feel. This is true also of all the muscles of the forearm. In the muscles of the left arm the same condition is noted as in the right. The right deltoid, biceps and triceps, even in the relaxed condition, feel firm and fibrous to the touch. The belly of the biceps feels as though it were made up of a bundle of coarse,

hard cords (giving a rope-like feel). The condition of the forearm is similar to the right. The muscles of the right thigh feel firm and fibrous. This is also true of the muscles of the leg, the sensation which is given to the finger being similar to that given by the arms. The condition of the left thigh and calf is practically identical with that of the right. The adductor and calf muscles, though relaxed, are, nevertheless, dense to touch, giving here and there the fibrous feel noticed in the biceps.

The face is smooth, the normal folds and wrinkles being somewhat obliterated; the surface of the forehead, nose, anterior aspect of cheek, temples and ear is smooth and glistening. The ears present a wax-like hue or gloss; indeed, they might pass as artificial models of the human ear in wax; this is especially true of the right ear. The lips are purple-pink in color. The vessels of the eyelids are much increased in number, so as to suggest a telangiectatic condition. Both cheeks are tinted a purple-pink hue, and, upon close examination, minute vessels are seen. The ears are tinted with the same hue, save in the prominent portions of the cartilage, such as the tip of the tragus and antitragus and the edges of the helix and antihelix, which are dead white.

The scalp and the neck, especially the back of the neck, reveal copious masses of epithelial scales or encrustations. These scales, the patient tells us, accumulate rapidly. They can, as a rule, be removed with difficulty both by washing and by gentle scraping, leaving a raw and slightly sensitive surface, probably the sensitive layer of epithelium, exposed. These patches are observed also back of the ears and at various parts of the chest and abdomen. The hands also present yellowish stains on palms and flexures of fingers, due to similar epithelial deposits. The backs of the hands and fingers reveal much pigmentation. The skin of the face is everywhere movable, though this mobility is restricted; this restriction is especially noticeable on the forehead. The scalp also is very restricted in movement, the loss of mobility being such as to suggest a very tightly drawn skull-cap. The fibro-cartilage of the ears seems denser, more resistant, and less flexible than normal. The cheeks are somewhat flattened. There is evident loss of superficial fat above and below the zygoma. The mobility of the facial muscles is well preserved. The skin of the eyelids, both upper and lower, seems somewhat thinner than normal. The eyelids and the sides of the nose at the root are slightly bluish in tint.

The neck and shoulders also present a glossy appearance. A number of telangiectatic patches are observed upon the

neck, shoulders, scapula and chest. The skin of the trunk is less smooth and more normal to the touch than that of the face and neck. Its mobility, however, is much diminished, and it is much firmer to the feel than normal skin. It is slightly more movable over the lower portions of the chest and abdomen than over upper portions of the chest. Tache cérébrale is noted freely over chest and abdomen.

The skin of the left thigh, especially in the middle third, appears to be slightly less mobile and more tense than normal. The middle and lower third of the thigh present the same yellowish encrustation of epithelium noted elsewhere. These



FIG. VII. Feet in case 2.

having been partially removed, the underlying skin presents numerous pink punctuate markings. The skin of the left knee, anterior aspect of the leg and foot, is also very much thinned and atrophied, and is highly glazed and shining. The skin over the foot is so thin that it permits the muscles and tendons beneath it to be plainly seen. The skin of the middle and lower third of the leg and of the dorsum of the foot presents a marked veining (the veins are apparently readily seen owing to the atrophied condition of the skin). The skin of the leg, especially in the lower portions, is much less movable than the skin of the thigh; it is especially tense over the dorsum

of the foot. The skin is especially thin over the first phalangeal articulation. The nails are much thickened, yellow, opaque and distorted; the matrix of the great toe is especially thick.

The right thigh is in every way similar to the left. Over the knee the skin is very tense, shining and thin. On the outer aspect of the leg and dorsum of the foot, the skin has the same appearance as in the left limb. The toes are glazed, and the nails are in the same condition as in the left. The atrophied changes of the skin also appear to be equally marked over the right foot, so that the tendons, veins and other structures can be readily seen through it.

The skin of the back of the neck, trunk and buttocks presents similar changes to those noted elsewhere, save that they are less marked. Its mobility seems lessened, and it seems denser to the feel than normal, but these factors are far less marked than in the skin over the anterior portion of the trunk. The soles of the feet are distinctly indurated, though less so than the palmar surfaces of the hands. There is no special loss of the plantar fat. An occasional fibrillary twitch, or twitch of a tendon, is noted in the hands. The palms of the hands present an indurated feel.

Pain is caused by attempts at passive movement or by turning the patient in bed, but this pain seems to be due to the strain placed upon the fibrous tissues and muscles, rather than to a strain upon the joints.

No sensory loss can be discovered, and, according to the patient's statement, none ever existed. The special senses are also normal. The growth of hair over the scalp, pubis and axilla and over the general surface of the body appears normal. The growth of beard is scanty. The patient swallows without difficulty liquid and semi-fluid food. No decided changes are noted in the vessels. The sound of the heart, however, is somewhat accentuated, while the pulmonary valves are distinctly roughened. No other visceral changes are observed. Bowels are constipated. Anal sphincter normal. The control over the bladder is slightly diminished; sometimes soils his bed before the urinal can be brought. The urine is normal in quantity. The solids are somewhat diminished, the urea, for example, varying from 230 to 270 grains daily. One specimen of urine revealed also the presence of peptone, though this observation was not repeated.

A blood examination by Dr. A. E. Taylor failed to reveal any evidence of leucocytic degeneration or karyokinesis.

The patient has never had sexual intercourse, has never had any seminal emissions; has occasional erections. Pubis is covered with a dense epithelial covering, similar to that noted on scalp and elsewhere.

When we analyze this case, we find that its clinical history, especially the mode of onset, accords closely with that frequently met with in chronic rheumatoid arthritis. The recurrent attacks of pain in the right knee, which appeared to be the starting point of the case, are exceedingly suggestive. Further, while trophic changes, involving the skin, nails and muscles, are not common in rheumatoid arthritis, they may occur. The case before us, however, is distinctly unusual in the extent to which these changes are present. We need only point to the tissues of the scalp, the fibro-cartilage of the ears, the tissues of the plantar, and especially of the palmar surfaces, all of which are indurated, much denser and less mobile than normal, and are evidently the seat of some diffuse sclerotic change. When we examine the muscles, the same fact is again apparent. As regards the muscles of the extremities, which are, of course, related to joints, it is conceivable that the changes found in them are arthritic in character. However, it is somewhat striking that the usual disproportion in the involvement of the flexors and extensors is not present. In purely arthritic muscular atrophy, the extensors suffer so greatly as to lead to marked flexion and contraction of the legs and arms; and, indeed, this is the usual position assumed in typical rheumatoid arthritis. Again, the diffuse induration of the muscles, very marked in certain situations, is a feature of the present case which must not be lost sight of; and even if we assume an arthritic origin for the muscular changes, how are we to explain the induration of the muscles which are not related to joints, such as the intercostals and the muscles of the abdomen? These, as pointed out, have an induration that is boardlike.

The deformity of the hands in this case also departs from that usually met with in rheumatoid arthritis. The so-called ulnar deflection is entirely absent. It is only in the toes of the right foot that abduction is seen. In the condition of the skin and nails, the hands and feet strongly

call to mind the sclerodactyle of scleroderma. Rigidity of the spine, so marked in this case, may also be met with in scleroderma, though much less pronounced. It was markedly present in a case of scleroderma described by me and reported in a former paper. In this instance the rigidity of the spine was not related to the skin of the back or of the neck, and appeared to be dependent upon involvement of the articular joints. In the same case, also, there was undoubted involvement of one shoulder joint without any involvement of the surrounding skin. Further, Legrange observed in sclerodactyle, loss of articular cartilage and calcareous deposits in the fibrous tissues, while Verneuil and Mirault observed short fibrous bands extending between the apposed joint surfaces in which there had also been destruction of the synovial membrane. While bone and joint changes do undoubtedly occur in scleroderma, and independently at times of the changes in the skin, such extensive joint changes as observed in the present instance, have not been observed in cases described as scleroderma. However, the case is extremely interesting as suggesting a general sclerotic process bringing about extensive changes in bones, joints, muscles, tendons and skin, similar in character to those seen in scleroderma. It would almost seem as though in scleroderma the process expended itself primarily upon other structures, while in this case, call it rheumatoid arthritis, if we may, the process had expended itself primarily upon the deeper structures. The study of this case suggests the further thought that under the name, chronic rheumatoid arthritis, are properly included two, if not more, clinical entities; one a disease in which a sclerotic process, similar to that which occurs in scleroderma, is active, and another in which the joint changes are attended by absorption of cartilage, eburnation of bone, osteophytic deposits, and secondary arthritic muscular atrophy.

FAMILY PERIODIC PARALYSIS.

With a Report of Cases Hitherto Published.

By EDWARD WYLLYS TAYLOR, M.D.,

Instructor in Neuropathology, Harvard University; Assistant in Neurology, Massachusetts General Hospital; Visiting Neurologist, Long Island Hospital, Boston Harbor.

(Continued from page 660.)

I find no reference since Goldflam's publication in 1897 bearing directly on this subject. The series of cases above outlined, which I believe to be complete, is extremely interesting from several points of view, and especially in this connection, first, because of the fewness of all cases, and, secondly, because of the relatively frequent occurrence of the affection in certain families. Cases inadequately reported I have not, in general, included in the foregoing summary, except to allude to them under the case or cases to which the individual author pays particular attention. For example, Cousot mentions five cases, and reports one completely; Goldflam mentions twenty-two, and reports six in detail.

A critical examination of all the cases included in the list given permits us, with reasonable certainty, to exclude a certain number from the typical form of paralysis already outlined.

The early cases of Cavaré, Romberg and Gibney may rightly be questioned, chiefly on account of their association with malaria. The very slow recovery in Gibney's cases would seem to place them in a distinct, though possibly allied, category. Fischl's case is anomalous, but probably belongs to the clinical group of the periodic family paralysis. The extremely interesting family history, as reported by Rich, must also be regarded as doubtful on account of the etiology, viz., exposure, the tonic

conditions of the muscles during the attacks, and the lack of information as to the reflexes and electrical conditions. Finally, Bernhardt's cases are, as he states, complicated by certain muscle changes of the dystrophic type. His patients suffered at all times from a distinct muscular weakness, although notably increased during the so-called attacks; otherwise, his cases are similar. We are justified in regarding the observations of Hartwig, Samuelsohn, Schachnowitsch, Westphal, Oppenheim, Fischl (doubtful), Cousot, Griedenberg, Pulawski, Goldflam, Burr, Hirsch, Bernhardt (with the above modification), and Taylor as the definite type of the condition under discussion. The number of cases reported by these observers is in all fifty-three. Of these, only sixteen have been described in any detail, the others occurring either in past generations, or in persons who were unavailable for a careful personal examination.

The second point is also of interest—that of the fifty-three cases, thirty-five occurred in three families, Cousot five, Goldflam nineteen, and Taylor eleven, the remaining eighteen being either sporadic, or associated with one other in the same family. From this fact alone the family character of the affection is sufficiently demonstrated.

Before entering upon a detailed analysis of this remarkable affection, it is as well to forestall a possible criticism in the use of terms by saying that in the present state of our knowledge, the word "disease" should be used with much caution as applied to this periodic paralysis. The tendency to dignify a symptom or a group of symptoms by calling it a disease entity has already been productive of much confusion of nomenclature in relation to affections of the nervous system whose pathology is obscure. As stated at the outset, the term "family periodic paralysis" is purely clinical and descriptive, and should be definitely so understood. If through a certain poverty of words we occasionally speak of the symptom-complex under consideration as a disease, it is merely as a matter

of convenience, and in no way as indicative of a dogmatic belief that a periodic paralysis, as such, can in itself represent a pathological entity. Our feeling, rather, is that we are dealing here merely with a symptom, or symptoms, of extraordinary constancy, whose etiology and pathological anatomy are as yet wholly obscure. This being understood, we may profitably attempt an analysis of the condition in the light of the reported cases and of our own, leaving the most subtle problems, viz., those of etiology and pathological anatomy to the end.

The first striking feature of the condition is that it has certain constant and cardinal peculiarities. It is characterized in its typical manifestation by its heredity, by its periodicity, by its confinement to the motor portion of the nervous system, by the electrical changes, and, finally, negatively by the practically perfect health of the individual in the free intervals. We shall consider each of these cardinal symptoms in detail.

Heredity: The affection is distinctly of a family type. In Cousot's cases five members of one family were affected; in Goldflam's nineteen, and in ours eleven. Schachnowitsch and Hirsch each report two cases as occurring in parent and child, and Goldflam again three in one generation. The other few cases are sporadic. In the condition described by Rich, twenty-two members of the family were affected, of whom fourteen were still living when his paper appeared. The two family histories of statistical interest are those described by Goldflam and Taylor. In Goldflam's family the predisposition was transmitted on the mother's side, through both male and female lines. It was not associated with other neuroses of any sort. Many of the family were entirely free. In my cases, also, the affection is on the mother's side, beginning with a male five generations back; from him transmitted to a son, thence to two daughters, one of whom had six children, three sons and one daughter, all but two being affected. Several of this generation are now living,

but I have been unable to secure any definite data. The other daughter of the previous generation had three children, one of whom, the mother of our patients, was affected. She, again, had three children, two of whom, a son and daughter (our patients), are sufferers from the condition, the other son having no trace of it whatever. It is, therefore, seen that in our cases the transmission has been absolutely direct through two females and two males. Those who have been exempt from the condition themselves have in no case transmitted it to their children. (Vide chart.) As regards other possible hereditary taint in the family, we find none of significance. One member in the second generation had epileptiform convulsions, but was not afflicted with periodic paralysis. The mother of our patients died of tuberculosis, and, possibly, their grandmother. To this fact we can attach no importance, as already stated. No other significant heredity appears.

In general it may be said that these cases have occurred in families of unusual nervous stability (See abstracts of histories); in no case do we find any evidence whatever of a degenerative family history. If a neurosis at all, it is distinctly not one of a degenerative type. The mental superiority of our family and its freedom from other nervous derangement is noteworthy.

Periodicity: A characteristic of the condition is its recurrence. The alternation of attacks of loss of motor power, with intervals of perfect health, is the peculiar feature which lends to the affection one of its chief interests. This periodicity is governed by no discoverable law, and varies greatly in different cases. From a condition of health, so far as the patient knows, he is more or less suddenly plunged into a state of helplessness, which, again, lasts a varying time, to give place in turn to a period of normal activity. Details of individual cases are not here necessary, inasmuch as the fact of periodicity applies to all, and is to be regarded as a fundamental characteristic.

Motor Paralysis: Another equally important and fundamental peculiarity is the absolute confinement of the affection to the motor sphere. Apart from certain doubtful subjective sensory disturbances, there is no evidence whatever to show that the sensory side of the nervous system is ever involved in the process. In our most carefully studied case there is no indication during the attack of the slightest sensory involvement. Tests for various sorts of disturbed sensation were wholly negative in result, nor was there ever any subjective complaint of pain or paræsthesia. It was noted at the examination made during an attack that strong faradic currents were borne without complaint, but this, we believe, was due rather to the failure of muscles to respond adequately than to an actual blunting of the feeling. This is so common in observation that further comment is unnecessary. It was furthermore observed that between the attacks there was small complaint of pain from strong currents. He is, perhaps, unusually tolerant of the peculiar sensations produced by electricity, as one frequently sees in non-neurotic persons.

In Case II., the sister, there is also no sensory disturbance, beyond a dull pain, following a severe attack, in the muscles affected. J. also complains at times of a feeling of soreness, which is evidently due to a natural local disturbance in the muscles themselves, following the paralytic stage.

The following vague disturbances of sensation have been described by previous observers: Hartwig speaks of a sense of numbness and formication in the paralyzed part, while finding objective sensibility unaffected. In Schachnowitsch's second case there were subjective sensory disturbances as precursors of the actual attack. In the Westphal-Oppenheim case there was an antecedent prickling sensation in the legs, and Oppenheim also noted a sense of prickling in the soles of the feet in the course of an attack, with otherwise perfectly normal sensibility.

Fischl, on the other hand, found in his case a distinct blunting of sensation in the affected areas, with, at times, an ice-cold surface temperature. This fact, in conjunction with the generally anomalous course of the case, a certain mental involvement, with lack of heredity, would seem to render it doubtful of classification, and we have so regarded it. Goldflam alludes to an annoying itching, chiefly between attacks, and also just previous to recovery from an attack. In Hirsch's case there were occasional sharp pains in one foot. In no case, excepting that of Fischl, was any objective change found, and the foregoing slight subjective disturbances we must regard as inconstant and unimportant.

The mind has in all cases remained unaffected, whether during or between the attacks. An occasional drowsiness has been observed, which in Fischl's anomalous case came to be practically an equivalent of the typical attack. The disturbance of the nervous system in these cases may, therefore, be regarded as essentially motor in its manifestation. This is a constant symptom. The distribution of the paralysis, however, varies in different cases, and at different times in the same patient. This will be considered in detail later. The paralysis is in all cases flaccid in type.

Electrical Changes: In general it may be said that there is a loss of direct and indirect electrical excitability, varying from absolute loss to both currents, to slight quantitative diminution, depending upon the completeness of the paralysis. Between the attacks the conditions have usually been normal or only very slightly abnormal. Exhaustive examinations have been made into the electrical peculiarities, chiefly by Oppenheim and Goldflam. Oppenheim found that at the height of the attack there was complete loss of reaction in certain groups of muscles, with an increased skin resistance. The return of reaction to faradism and galvanism was gradual, like the return of motor power. There was always quantitative diminution

during attacks. Response, when obtainable at all, was quick. No R. D. The results of Oppenheim's electrical examinations were not entirely uniform. Later observations on the same patient showed a slight atrophy of those muscles which had the poorest reactions between the attacks.

Goldflam also has examined his cases with extreme thoroughness, and with interesting results. In addition to the quantitative changes, up to complete loss, observed during attacks, Goldflam describes certain changes in the free intervals, which he regards as of the utmost significance and importance. In two of his cases, brothers, he found the direct and indirect irritability markedly diminished to both currents between the attacks, associated with rapid fatigue. The character of the contraction was also changed; it was slow, and there were, in addition, various polar changes. Partial R. D. was obtained and both Ca. C. Te. and An. C. Te. with weak currents. He found qualitative changes more prominent in the free intervals, and quantitative, often up to complete loss, in the actual attacks. Other observers have all noted the quantitative diminution of electrical excitability during the attacks, but have in general found the condition between normal in this, as in other respects. With the possible exception of Oppenheim, however, no one has so thoroughly investigated the matter as Goldflam. In our own case a quantitative diminution to faradism, which it was alone possible to use, during the attack was pronounced. Noteworthy in our case was a quantitative diminution in the facial nerve and its distribution. Between the attacks we could demonstrate absolutely no polar changes whatever. Response was also quick and normal to faradic and galvanic currents. There was an apparently increased skin resistance, alluded to by Oppenheim as occurring during the attacks. The only noteworthy peculiarity in our case was the failure of reaction of the right peroneal group of muscles. In the light of

Goldflam's researches, this fact may be of more significance than we are at present disposed to grant. Future examination will demonstrate its importance, if it have any. Considering the fact that all other muscles and nerves tested responded normally, we would reserve judgment of the unexplained failure of this one group.

The general changes regarding the electrical changes may be summed up by saying that there is a loss or marked quantitative diminution during attacks, and considerable evidence of a disturbed neuromuscular excitability in the free intervals, not yet demonstrated as constant.

Reflexes: Hardly less noteworthy than the electrical changes is the behavior of the reflexes. In all cases, when any mention is made of the matter, the deep reflexes, of which the knee-jerk is the type, have been reported lost during the height of the attack, and present in the intervals. This was verified in my case. The same holds true, in general, of the peripheral reflexes, though not to the same degree. Oppenheim reports: Plantar, lacking; cremaster and abdominal, present; Hirsch: Peripheral reflexes lost, excepting abdominal, which were weak; Taylor: No plantar; cremaster, slight; abdominal and epigastric, active. Pulawski and Goldflam, on the other hand, report cutaneous reflexes present. The disturbance of the reflexes depends apparently wholly upon the extent of the paralysis. Inasmuch as the legs are most often and most completely involved, we find an explanation of the constant loss of knee-jerks, and the greater frequency of the loss of the plantar than the other cutaneous reflexes. With our relative lack of knowledge of the peripheral reflexes, nothing further is to be said. Our chief interest centres in the knee-jerk.

The Free Intervals: We have maintained that the patients suffering from this affection are normal between the attacks. This is, in general, true, but the rigid examinations to which certain of the cases have been

subjected would seem to throw doubt upon its absolute acceptance. In our cases nothing abnormal was found; the patients themselves feel and, so far as they know, are perfectly well and strong, excepting at the time of the attack. This has been the usual verdict. Oppenheim, however, found slight atrophy of the hand muscles in his case, and later weakness of the legs, associated with constant defect in electrical reaction. Goldflam describes in detail electrical abnormalities between the attacks, and definite changes in the muscular system, upon which he bases his theory of the affection. Bernhardt, whose cases we have considered somewhat atypical, found muscle changes, which he regards as allied to the dystrophies, from the beginning of the affection or even before. With these exceptions, the general statement stands.

In the foregoing discussion we have pointed out what may be looked upon as the cardinal symptoms of the family periodic paralysis. These symptoms are so constant that their absence would rightly throw doubt upon the diagnosis. They are: Periodicity, flaccid motor paralysis, loss of electrical excitability, loss of deep reflexes, and, between attacks, relatively perfect health.

Other symptoms vary somewhat in different cases, and for this reason demand a painstaking analysis. We shall attempt to describe them in as systematic a manner as possible, omitting for the time being any discussion of possible causes.

Attack; Prodromata: Various symptoms have been described as occurring immediately antecedent to an attack, though none of them are constant in all cases. Hartwig's patient had a feeling of weariness, with some sweating and considerable numbness and formication. Westphal-Oppenheim noted heat and sweating, with a desire to urinate, which could not at once be satisfied. Fischl's (doubtful) patient had headache, backache, poor appetite, somnolence, rapid pulse, with a normal surface temperature of the head, body and arms, and an icy-cold temperature of

the legs, associated with great pallor, suggesting a vasomotor disturbance of a violent sort. Cousot found feebleness of articulation and a desire to walk. Goldflam, in his best observed case, alludes to the fact that the attacks usually came on Friday, no doubt a coincidence; that the patient had itchings on the well evenings, but none on the evenings preceding the attack; that he was capricious as to food, and was later annoyed by a feeling of coldness in the legs and by occasional thirst. In my best observed case there are no prodromata whatever, excepting a feeling of weakness, which is rather a part of the actual attack than a warning of its onset.

Evidently, then, these prodromal symptoms are exceedingly inconstant. Sweating and thirst are most frequent, but they are much more often absent than present. The fact that the attacks come on so frequently at night may explain the fewness of the prodromal symptoms.

Age of Onset: The age at which the attacks usually begin varies within somewhat narrow limits. The data on this point, so far as obtainable, are as follows: Cavaré, before twenty-one; Hartwig, at twenty-three; Samuelsohn, at eighteen; Westphal, at twelve; Fischl, at eight; Cousot, four cases at fourteen, nine, ten and ten, respectively; Griedenberg, before twenty-one; Pulawski before twenty-one; Goldflam, case chiefly studied, at thirteen. The mother of Goldflam's case had but one attack, at thirty-six. Goldflam also refers to a number of young children similarly affected, who are said to have died during attacks. Burr's case began at ten, Hirschs at the nineteenth or twentieth year, and Taylor's first case at fourteen or fifteen; the second at twelve. Of the other nine cases in the family, the tradition is that the attacks began always at or about the time of puberty. The latest case was at eighteen.

From these statistics, it will appear that the affection is evidently one that owes its origin to certain con-

ditions prevailing in youth and the developmental period of life. With the exception of a few cases mentioned by Goldflam, the onset has always been before the twenty-fourth year, and, in the great majority of best reported cases, it has been between the tenth and twentieth years of life. Of those reported under the tenth year, Fischl's is very doubtful, as before stated. Cousot's was imperfectly reported, as also Goldflam's cases occurring in childhood.

Sex: In general there seems to be no discrimination as to sex, though it happens that in the carefully reported cases there are but three females—reported by Fischl, Goldflam and Taylor. The affection is transmitted, however, equally through the female line. Of the eleven cases in Taylor's family, five were women.

Muscles Involved: The most constant feature in this regard is the paralysis of the lower extremities. In the typical cases the legs are always affected, usually first and most severely. Body and arms are later involved in the severe attacks, as are also the neck muscles. Very unusual is an involvement of any of the cranial nerves. An analysis of the observed cases shows certain variations from the general rule. Burr, for example, found in his case a hemiparesis, not, however, involving the cranial nerves; ordinarily, the paralysis was bilateral. Hirsch, in one attack of his patient, speaks of the weakness beginning in the arms. In Romberg's case and Fischl's cases the attacks were practically limited to the legs, as they were at times in Griedenberg's case. Cousot noted feebleness of articulation as an early symptom, with difficulty later both in speech and deglutition. The usual description, however, is: Legs, arms, body, head involved; cranial nerves free, coughing and sneezing often impossible; at times superficial respiration. With slight variations these were the conditions found by Hartwig, Samuelsohn, Westphal-Oppenheim, Pulawski, Goldflam, Hirsch and Bernhardt. In our own case the paralysis is unusually complete. Legs,

body, arms, neck muscles are always involved in these severe attacks. Most noteworthy, however, is the observation that the facial nerves are occasionally included, to a less degree, and in the case of Mrs. G. (Case II.) the motor branches of the trigeminal nerves are slightly affected in the most serious attacks. In her case, also, a threatening dyspnœa, alluded to also in Goldflam's second case, has occurred in her worst attacks.

The distribution of the paralysis is sufficiently constant to justify the general statement that its greatest severity is in the leg muscles, and that it gradually grows less the higher on the body we go, the ocular nerves, for example, never being involved, and, so far as we know, the fifth only in our one case. The involuntary muscles are not affected.

Order of Involvement of Muscles in Attack: We have already, in a preceding paragraph, indicated that the legs are first affected, and later the other portions of the body in a somewhat regular sequence. The exact determination of this point in our case and in others is difficult, owing to the fact that the paralysis usually comes on during sleep, and the patient finds himself on waking completely paralyzed. My patient J., for example, could give me no definite information as to which muscles were first affected. His general impression, however, is that the legs first grow weak.

Mechanical Irritability of Muscle and Nerve: Goldflam lays stress upon the fact that the mechanical nerve-muscle irritability is markedly changed during an attack. Irritability of the muscles through tapping or pressing a nerve, in his case, was often totally lost. The idiomuscular irritability was altered in such a way that the fascicular contractions in the muscles were lost, and later the transverse swellings occasioned by the blow also disappeared. The return of the mechanical irritability of the muscles and nerves he found antedated somewhat the return of electrical excitability. Goldflam regards this observation

as of great importance, though, naturally, secondary to the still more remarkable electrical changes. Hirsch speaks of the idiomuscular irritability being less than the normal in his case.

Order of Recovery: It appears from a study of the cases that the order of return of power is the inverse of the onset. As a rule, recovery begins in the upper extremities, and extends later to the legs.

Rapidity of Onset and Recovery: The usual rule is, so far as it is possible to observe, a gradual onset, usually of several hours, at times of several days, before the height of the paralysis is reached; a stationary stage of varying length, and an equally gradual recovery in the order named. A sudden loss of power and a sudden recovery are exceedingly rare. Hirsch reports a sudden recovery, and also Fischl, from one attack, and Goldflam a sudden onset.

Time of Onset: It is usually at night. Schachnowitsch, Westphal-Oppenheim, Cousot, Pulawski, Bernhardt and Taylor report cases in which the patient wakes paralyzed. This is always so in my best reported case. The other case, Mrs. G., has attacks not infrequently which begin during the day.

Length of Attack: This varies within very wide limits, from less than an hour (Fischl) to a week (Burr). The usual duration of a well-pronounced attack is from ten to forty-eight hours. The length varies greatly in the same patient at different times. It is a common experience for my patient J. to be unable to leave his bed before 11 or 12 o'clock, and hardly less common for him to be helpless until evening. (Vide letter). He also has frequent feelings of weakness, which may hardly be called actual attacks. These feelings are rather precursors of the most serious ones, which are sure to come. In general, the longer the attack, the more complete the helplessness and the more profound the reflex and electrical changes.

Frequency of Attacks: Nothing definite is to be said on this point. Goldflam speaks of a case in which but one

attack occurred during life, and that in the thirty-sixth year. In one of my cases the attacks occur at times daily, and frequently several times weekly, with intermissions. Cavaré and Romberg spoke of a quotidian type, which they associated with malaria. Samuelsohn's case had two attacks a year for a while; Hirsch's, one yearly for a certain period; Westphal's, one every four and six weeks; Goldflam's, weekly to yearly. There is in this absolutely no constancy. It is to be observed, however, that each individual has a certain type of attack, which may vary greatly in degree, but not markedly in kind. Since the onset of the affection in my case J., scarcely a week has passed without some intimation of its presence. His sister, on the other hand, is entirely free for months at a time.

Total Length of the Affection: Although, for obvious reasons, definite information is somewhat uncertain on this point, yet the stated or implied belief of the various writers on the subject is that the attacks continue unchanged and unchecked through life. There is no intimation that they grow less or cease. Of very particular interest, then, is the statement made in regard to my family, that in all the cases the attacks have tended to grow less with advancing years, and finally to disappear entirely between forty and fifty. There are at present members of the second generation living who have had, but are now practically free from the attacks. In view of Goldflam's and Bernhardt's theories, later to be discussed, this fact must be of great importance.

Abortive Attacks: There is no adequate evidence to show that an attack is ever actually aborted. Schachnowitsch, Goldflam and Taylor found that active muscular exercise had the effect of postponing an attack. This is very striking in my case. A feeling of weakness may be dispelled by walking, for example, but he is sure that it is merely temporary, and that he pays all the more dearly, perhaps the next day, for his temporary escape. A relapse before recovery has entirely taken place may occur,

as shown in my case, described in detail under his personal history. This is unusual.

Equivalents: The only instance we have been able to find of what may properly be called an equivalent is that described by Fischl. In place of the paralytic attack, his patient, a child of eight, developed a peculiar drowsiness, which came on in paroxysms, from which he finally fully recovered. The case is in general too uncertain to render the observation, in this connection, of value.

Heart: Oppenheim was the first to call attention to a remarkable temporary affection of the heart. His observations were later verified by Hirsch. Oppenheim found a slight but definite enlargement, with murmurs indicative of mitral insufficiency, during the attack, which quickly gave place to normal conditions when the paralysis disappeared. Goldflam describes the heart as arrhythmic in action at times, with a weak, blowing systolic murmur at the base and a slight accentuation of the second sound, combined with a slow pulse, and no other evidence of circulatory disturbance. This condition, however, excepting the arrhythmia, persisted in the free intervals; there was no enlargement of the heart, and, in general, the disturbance could not be attributed to the direct influence of the attack, unlike Oppenheim's case. In view of a later case, Goldflam has somewhat modified his view. Hirsch found almost precisely the same condition that Oppenheim had—area of dullness enlarged, first sound at apex impure, slight accentuation of the second pulmonic, insufficiency of the mitral valve; pulse regular, 78. The morning following the attack, the heart was practically normal. Up to this time I have not been able to find any temporary heart abnormality in my case. To determine the point positively, however, repeated examinations in a large number of attacks should be made.

The pulse shows no noteworthy change during the attacks, and, in general, it may be said that, apart from a temporary heart dilatation noted in two cases, not much

is to be learned from a study of the circulatory apparatus.

Temperature: Nothing noteworthy. Changes in superficial temperature were observed in Fischl's doubtful case.

Bladder and Rectal Functions: It is an interesting observation that in my case the desire to pass urine or fæces is abolished during the attack, even if of thirty-six hours' duration. This is never due to a true retention, but to a lack of desire. The patient himself explains it on the ground of his helplessness, and the consequent discomforts of either act. This evidently is hardly a sufficient explanation. Probably somewhat less urine is secreted than under normal conditions, and certainly much less water and food are ingested. (See previous remarks on appetite and thirst.) This, with the general atony of the muscles, no doubt brings about the condition; it is not incapacity, but, rather, lack of necessity. Westphal speaks of a desire to urinate as a preliminary symptom, with a temporary inability to do so. With this trifling exception, we find no mention of disturbance of the rectal and bladder functions. When the matter is mentioned at all, the writers speak of the condition as normal and the sphincters as uninvolved.

Other symptoms alluded to in the reported cases are too infrequent and inconstant to require individual consideration.

It will be impossible and undesirable to draw a sharp line between the pathological anatomy and the etiology of this affection. For the sake of clearness of exposition, however, we shall attempt to consider these two final matters under separate headings.

Pathological Anatomy: There are no constant changes in any of the internal organs; the only temporary change as yet described is a dilatation of the heart during an attack (Oppenheim, Hirsch). The spleen has shown no alteration between or during attacks. Inasmuch as death from this cause is practically unknown, no minute post-

mortem examination of the nervous system has been made. Nor can we suppose any constant lesion of the nervous mechanism, demonstrable by the microscope, could, under most favorable conditions, be found, since intervals of health, certainly so far as the nerve mechanism is concerned, alternate with the typical attacks. Leaving aside for the moment the etiology, it is evident that the chief interest of the affection, from the point of view of pathological anatomy, must centre in the muscular system, although certainly no adequate explanation of the origin or nature of the attacks is to be sought in the muscles themselves. Excision of a small bit of muscle has been practiced both by Oppenheim and Goldflam, who have arrived at quite contrary opinions as to the significance of their findings. Oppenheim describes a bit of muscle, excised from the deltoid, as showing a waxy degeneration, which he regards, however, of most doubtful pathological significance. He attaches, in fact, no weight whatever to the observation. Goldflam, on the other hand, from a study of muscles from several of his cases, using muscle from a cadaver as control, comes to the conclusion that the pathological changes in the muscle are of extreme importance, and offer at least a partial explanation of the clinical picture. He found an increased diameter of the individual muscle fibres, a general hypertrophy of the fibres, a rarefaction of the primitive fibrillæ and vacuole formation. To these muscle changes Goldflam attributes the persistent electrical anomalies, which he alone has found constant between the attacks. Such changes, or any permanent changes whatever, manifestly do not explain the periodic paralysis. Oppenheim's and Siemerling's⁹ investigations would indicate that in so far as Goldflam drew conclusions by comparison with muscle from the cadaver he was in error. This point Goldflam, in general, admits, but feels none the less confident that

⁹ Oppenheim-Siemerling: *Centralbl. f. med. Wissenschaften*, 1889, pp. 705, 737.

definite pathological changes were present. In his last article Goldflam reiterates the ideas previously expressed.

Beyond this we have no knowledge as to the condition of the muscles. Bernhardt's cases are interesting in this connection, inasmuch as his patients from their earliest years had constant difficulty in the use of certain muscles, entirely apart from the periodic attacks. For this reason, because of the almost undoubted muscle changes in Bernhardt's cases, as he supposed of an allied sort to the various dystrophies, we have hesitated to include them among the uncomplicated forms of so-called family periodic paralysis. The justification for this may seem doubtful in the light of Goldflam's researches, but we have been led to this standpoint for the reason that in our family recovery from the attacks always occurs at about the fiftieth year. If a dystrophy, which both Goldflam and Bernhardt supposed, were at the bottom of the difficulty, it is not easy to explain this fact, which appears absolutely undoubted in our family. As before stated, the changes described by Goldflam may be an accompaniment but can in no way be regarded as causative of the periodic attacks, which, after all, constitute the so-called disease. It will be remembered, also, that Oppenheim found a slight muscular atrophy in the hand muscles, but apparently does not take the position that the affection is to be classed with the muscular atrophies or dystrophies, Goldflam does not hesitate, however, to say that through his researches on the muscle he has placed the affection in the category of the organic diseases. His own words are: "Durch diesen Muskelbefund ist die paroxysmale Lähmung aus der Reihe der Neurosen geschieden, sie tritt in das immer sich erweiternde Gebiet der auf organischen Störungen beruhenden Erkrankungen, speciell muss sie in die grosse Kategorie der bereits bekannten familiären (mit gleichartiger Vererbung) Erkrankungen eingereiht werden, als welche die Dystrophia muscularis progressiva, die neurotische Muskelatrophie Hoffman's, die Fried-

reichsche Krankheit, die Myotonia congenita zu nennen sind."¹⁰

Bernhardt is inclined to the same opinion; others would, as yet, not be so dogmatic.

As to the location of the process, whatever it may be, which produces the paralytic attacks, we may certainly limit it to the spinal motor neuron and its associated muscles. Westphal suggested, as an explanation, a periodic disturbance of circulation in the cord. Were such a circulatory disturbance the cause, we should have to suppose a limitation of its action to the anterior horns, since sensation is not affected. This is evidently a mere assumption, and also, in the light of later researches, does not explain the observed facts. Oppenheim has suggested a similar circulatory disorder in the periphery, which certainly may exist, but is difficult to prove. What evidence we have goes to show that the periodic disturbance is peripheral, and probably associated with the terminal distributions of the nerves in the muscles, and almost undoubtedly with the muscles themselves, as shown by the loss of myotatic, as well as nerve, irritability. Were it necessary to find a single point of disturbance, we should locate it in the muscle rather than in the nerve or its endings. It is, however, altogether possible, and incapable of refutation, that it may be the result of a temporary lesion in both muscle and nerve.

The *blood* shows certain changes of interest in the few cases in which it has been examined. In my case Dr. H. F. Hewes has twice studied the blood, both specimens being taken during the free interval.¹¹ The details of his reports have already been given under the clinical history. In general, he found a lymphocytosis, with no evidence of anæmia; a large percentage of basophiles, which he regards as characteristic of a reduced condition

¹⁰ Goldflam: Loc. cit. Ref. 18, p. 27.

¹¹ Unfortunately it has, as yet, been impossible to make an examination of the blood and urine taken during the attack.

of nutrition. The count, he states, would be normal for a child of ten, but not for a man of twenty. Dr. Hewes finds also a relatively small number of white corpuscles.

Goldflam alone of the other observers reports a blood examination. Klein, who made the examination found a constant but varying leucocytosis during the attack, which was normal in the intervals. Noteworthy is the fact that Klein also found a definite lymphocytosis, 40 per cent., in the free intervals, which is identical with Hewes' observation. In the free interval eosinophiles often show an increase to 5 per cent. In the attack there was a marked neutrophile leucocytosis, with a reduction in the number of eosinophiles. The blood, therefore, in both Goldflam's case and mine shows distinct pathological qualities. We may not, however, draw deductions from this fact; it is quite impossible for us to say in what relation the blood changes stand to the paralytic attacks. It is, however, probable that both are due to a common cause, of which we have as yet absolutely no definite knowledge.

The ordinary routine examination of the *urine* in the various cases has shown nothing, excepting in Goldflam's later cases, in which traces of albumin and pathological cells were found during an attack. Goldflam again was the first to make a detailed study in order to determine, if possible, the presence of a toxic substance, to which might be attributed the periodic attacks. His efforts were, in part, successful. The analysis was made by Knaster, and resulted in the isolation of certain substances whose toxic effect, however, could not be demonstrated on animals. A rabbit, for example, injected with the supposed ptomaine, died, but with none of the symptoms of the periodic paralysis, as seen in man. The injection of the urine itself seemed more toxic when the urine was taken during an attack than in the interval. These investigations must be regarded as, in general, negative, and certainly show nothing as to the pathology of the affection. The examination of a twenty-four-hour specimen

of urine in the free interval in my case suggested a somewhat low condition of metabolism. The excretion of urea was below normal for a person of his age. The twenty-four-hour amount was also low. Otherwise, the urine was essentially normal. (Details are given under clinical history.)

Examination of the stools has thrown no light on the question. It has not been made in my case, and there is no evidence that it has been done by any observer with anything approaching adequate thoroughness.

Etiology: The exciting cause of these periodic attacks has been up to this time absolutely elusive, and no doubt will remain so until our methods of investigation are much more highly perfected than now, and until we are able to distinguish clearly causes from effects, and concomitant conditions from both. There is evidently a striking hereditary predisposition in the affection, which, naturally, in no way helps to explain the exciting cause. The association of the attacks with malaria led several of the earlier writers (Hartwig, Cavaré, Gibney) to bring the two conditions into casual relationship. Apart from the doubt which attaches to the classification of certain of the cases, notably Gibney's, it would appear that the mere fact of periodicity possibly led to the idea of malaria as a cause, an error from which we are not yet wholly free. Now that we are able to demonstrate the malarial organism, there is no possibility of the perpetuation of this error. The organism has never been found in cases of periodic paralysis, and as our clinical knowledge has grown, it has become more and more evident that the condition has no affinity to malaria beyond the superficial fact of its periodicity.

An observation which we have made in common with others is that the attacks almost invariably occur at night, and, what is more important in this connection, that they are usually preceded by a day of exceptional muscular activity. My patient is able, with a certain degree of as-

surance, to predict an attack from his previous day's employment. A long bicycle ride on a tandem, in which he was obliged to do most of the work, led to one of the worst attacks the following night and day he has ever had. Such experiences are common with him, though not absolutely invariable. Not less interesting is the fact that the attacks never come on while he is exercising, a common observation of others as well, but only after he has rested and, preferably, slept. Our conclusions from these relatively constant facts must simply be that muscular exertion, followed by muscular rest, is the condition of an attack.

This statement of the fact, however, brings us no nearer the actual exciting cause. Bernhardt, Oppenheim, and especially Goldflam have urged the probability of an autointoxication of some kind, the conditions of whose activity are given by exercise, followed by rest. Van Gieson¹² in his recent paper expresses a like opinion, and certainly, to us, no other cause seems adequate. In this case we must suppose that, depending upon a family peculiarity in certain stages of metabolism a poison is generated within the system, whose action is paralyzant to peripheral motor nerves and muscles. The analogy to curare has been suggested by Bernhardt. It is interesting, but not altogether apt, inasmuch as the effect of curare is confined to the nerve, whereas in the periodic paralysis the toxic agent, whatever it may be, leads to a loss of myotatic irritability as well. Further speculation on this point is unprofitable. We, as yet, have no demonstrative evidence of the theory of autointoxication. Nevertheless, it remains by all means the most plausible working hypothesis. The source of origin of the problematic poison is wholly obscure. In the hope of finding some light, careful investigation as to the digestive functions has been made, with no result. It is quite impossible in

¹² Van Gieson: *The Toxic Basis of Neural Diseases*, State Hospitals Bulletin, 1896, I., No. 4, p. 470.

my case or others to logically trace an attack to a dietary error, or to a possible retention in the body of faecal matter. The lack of appetite during attacks cannot be looked upon as of the slightest significance in this connection.

For obvious reasons syphilis, neuritis and hysteria play no part in the etiology. Emotions have, in certain cases, seemed to have some effect in the production or checking of an attack, but not to a significant extent.

The results of pathological and etiological investigations hitherto made are, therefore, positive as regards actual muscular changes (Goldflam, Bernhardt) in certain cases, and entirely negative as regards the nature of the substance, if such it be, which causes the periodic attack. The pathological changes in muscle and, possibly, in blood and urine are interesting as facts, but not as explanation.

Diagnosis and Classification: The affection presents no difficulty in diagnosis when occurring in a typical form. The combination of symptoms is unique and unmistakable. Goldflam has in one case of Landry's paralysis temporarily made a diagnosis of paroxysmal paralysis.

The classification of the disturbance offers much greater difficulty. As already indicated, Goldflam and Bernhardt are strong in their belief that the affection should be classed among the dystrophies as an organic disease of the nervous system, giving as an argument that changes (Goldflam) are found in the muscles. Admitting this fact, however, the periodic paralytic attacks, which alone are significant, remain totally unexplained, inasmuch as most dystrophies occur without such attacks. Goldflam admits to a certain extent the inconsistency, but retains his somewhat dogmatic position. In our opinion, and particularly in our family, we feel altogether justified in subordinating any possible muscular changes, and fixing our attention solely on the attacks, which are the disease, as clinically observed.

To our mind, a more interesting and instructive analogy is that between this affection and the so-called

congenital myotonias. Although differing widely in manifestation, the two affections at least have this in common, that they occur paroxysmally.

In this connection a reference to Rich's cases is of interest. It will be remembered that in his cases cold was invariably the exciting cause of the attack, but that the attack was always of the character of a tonic spasm. In this curious family history we no doubt have to do with what Eulenburg has described as *Paramyotonia Congenita*, a disturbance allied to myotonia of the ordinary type. Further study of these spasmodic affections of the neuromuscular system in relation to the flaccid periodic paralysis will no doubt yield much of interest. To ally the conditions under consideration with the muscular dystrophies is surely a begging of the real question at issue. Goldflam, however, states that the periodic paralysis has the closest analogy with congenital myotonia, but our knowledge of the pathology of myotonia is evidently too vague to admit of dogmatic statement. That periodic paralysis is to be included in the general etiology of the hereditary affections goes almost without saying, but we are overstepping our knowledge when we attempt too closely to force its relationship to any one.

Prognosis: It is usually good so far as life is concerned. Apart from young children, but two deaths have been reported, one of doubtful significance, the other apparently induced by a therapeutic attempt. My family has the peculiarity of showing a tendency to recover in middle life. Others reported have remained unchanged through life, so far as observed.

Treatment: Absolutely unavailing. It is evident that a successful treatment is not likely to be instituted until a cause is found; drugs, including quinine, in the undoubted cases have been entirely inefficient.

The affection discussed in the foregoing pages evidently offers one of the most subtle problems with which medicine has to deal. The clinical phenomena are ex-

traordinarily well defined; the pathological anatomy is vague; the etiology is absolutely obscure. The solution of the problem already reaches far beyond the field ordinarily occupied by the neurologist; a laborious investigation into the chemistry of the metabolism of so peculiarly predisposed a series of individuals may finally bring the desired solution. At present, excepting that the problem is more definitely stated, we are not further advanced than was Westphal, more than ten years ago, when he said: "Wir stehen somit dem geschilderten Krankheitsfalle als einem Räthsel gegenüber."

BIBLIOGRAPHY:

(1) Hartwig: Ueber einen Fall intermittirender Paralysis spinalis. Inaug. Dissertation, Halle, 1874. Ref. by Bernhardt, *Centralbl. f. d. med. Wissensch.*, 1875, No. 26, p. 428.

(2) Samuelsohn: Fall von intermittirender Paraplegie. *Verein für wissenschaftliche Heilkunde zu Königsberg*, 1. Pr., Oct. 13, 1876. *Berl. kl. Wochenschr.*, 1877, xiv., No. 41, p. 607.

(3) Schachnowitsch: *Wratsch. Russisch.*, 1882, p. 537. Ref. in Goldflam's paper, 13, of Bibliography.

(4) Gibney: Intermittent Spinal Paralysis of Malarial Origin. *Am. Jour. of Neurology and Psychiatry*, 1882, vol. i., p. 1.

(5) Westphal: Ueber einen merkwürdigen Fall von periodischer Lähmung aller vier Extremitäten, mit gleichzeitigem Erlöschen der electrischen Erregbarkeit während der Lähmung. *Berl. kl. Wochenschr.*, 1885, No. 31, 32, p. 489.

(6) Fischl: Ueber einen Fall von periodischer auftretender Lähmung der unteren Extremitäten. *Prag. med. Wochenschr.*, 1885, No. 42, p. 401.

(7) Nachtrag zu dem Aufsätze "Ueber einen merkwürdigen Fall von periodischer Lähmung aller vier Extremitäten," u. s. w. *Berl. kl. Wochenschr.*, 1886, No. 11, p. 165.

(8) Cousot: Cas de paralysie périodique. *Bul. d'Acad. de Méd. de Belgique*, 1886, No. 7.

(9) Cousot: Paralysie périodique. *Rev. de Méd.*, 1887, vii., p. 190.

(10) Griedenberg: *Wratsch. Russisch.*, 1887, p. 930. Ref. in Goldflam's paper, 13, of Bibliography.

(11) Goldflam: Ueber eine eigenthümliche Form von periodischer, familiärer, wahrscheinlich auto-intoxicatorischer Paralyse. *Wien. med. Presse*, 1890, No. 36, 37, 38, 39, p. 1418.

(12) Pulawski: La paralysie totale des quatre extrémités et du tronc durant 48 heures. *Gaz. hebdomadaire de méd. et de chir.*, 1890, No. 48, p. 570.

(13) Goldflam: Ueber eine eigenthümliche Form von periodischer, familiärer, wahrscheinlich auto-intoxicatorischer Paralyse. *Zeitschr. f. klin. Med.*, 1891, xix; Supplementary vol., p. 240.

(14) Oppenheim: Neue Mittheilungen über den von Prof. Westphal beschriebenen Fall von periodischer Lähmung aller vier Extremitäten. *Charité Annalen*, 1891, xvi., p. 350.

(15) Burr: Periodic Paralysis with the Report of a Case. *Univ. Med. Mag.*, Phila., 1892-'3, p. 836.

(16) Hirsch: Ueber einen Fall von periodischer, familiärer Paralyse. *Deutsche med. Wochenschr.*, 1894. No. 32, p. 646.

(17) Rich: A Unique Form of Motor Paralysis due to Cold. *Med. News, Phila.*, 1894, lxx., p. 210.

(18) Goldflam: Weitere Mittheilung über die paroxysmale, familiäre Lähmung. *Deutsche Zeitschr. f. Nervenheilk.*, 1895, vii., p. 1.

(19) Bernhardt: Notiz über die familiäre Form der Dystrophia muscularis progressiva, und deren Combination mit periodisch auftretender, paroxysmaler Lähmung. *Deutsche Zeitschr. f. Nervenheilk.*, 1896, viii., p. 111.

(20) Goldflam: Dritte Mittheilung über die paroxysmale, familiäre Lähmung. *Deutsche Zeitschr., f. Nervenheilk.*, 1897, xi., p. 242.

DISCUSSION.

Dr. Wharton Sinkler, in connection with the paper of Dr. Taylor, wished to place on record a case which came under his observation last summer. The patient was a man with facial paralysis, who gave a history of five previous attacks in the course of about ten years. Each of these lasted several weeks, and ended in complete recovery. No satisfactory cause for the attacks could be elicited.

Dr. C. L. Dana had seen a number of cases of periodic paralysis; none, however, of the family type. From his observation of these cases, including the one reported by Dr. Gibney, he had come to the conclusion that, while some of them were cases of recurrent poliomyelitis, the majority were purely hysterical in character. The speaker expressed the opinion that the question of periodical paralysis due to malaria was still unsettled. He had never seen any cases of that kind, and in several instances where patients were sent to him with that diagnosis, the paralysis proved to be simply functional or hysterical.

The family type of the disorder reported by Dr. Taylor was extremely interesting. It was certainly rare, and, perhaps, a little light might be thrown upon the subject by studying allied conditions. Dr. Dana said he had seen a number of patients who had informed him that when first waking in the morning they were unable to move for half an hour, or even longer. They suffered from waking palsy and a certain amount of waking numbness. Cases of ptosis are not uncommon, and the same is true of cases of morning sweating, where the patients become bathed in profuse perspiration just as they awaken. Other peculiar morning disorders are known, and perhaps these palsies may be in the same category. Dr. Dana said he had always regarded them as the result of diathetic disturbances. They usually occur in persons of the neurasthenic or lithæmic type, and in the former they are probably associated with a congenital disturbance or defect of the nervous system. The ordinary types of morning pa-

ralysis seem to be the result of some toxic condition, associated with an asthenic condition of the nervous system.

Dr. Joseph Collins had observed three cases of periodic paralysis; none, however, of the family type. One of the cases had been reported by him under the title of asthenic bulbar paralysis, and it was a typical example of that affection. When Dr. Collins first saw her, her symptoms were those of profound neurasthenia. About two years after the commencement of her illness she had double ptosis, double facial paralysis, paralysis of the extremities, more marked in the upper, and ballooning of the abdomen; her general condition being very similar to that of surgical shock, and indicating an overwhelming disorder of the sympathetic nervous system. After a few days' duration the paralytic manifestations disappeared. Sensory disturbances were never noted. In the course of two or three years she had had about five attacks similar to this one, and in the interval the symptoms of asthenic bulbar paralysis persisted.

The second case was a woman who had been referred to him by Dr. Fordyce. She had complete motor and sensory paralysis on one side, and the hand and forearm, as well as the foot, of the same side showed the manifestations of Raynaud's disease and erythromelalgia. The symptoms came on in attacks, and had so far recurred three times.

The third case was one seen in Dr. Dana's clinic some years ago. The patient was a boy of fourteen, with a profoundly neuropathic family history, who had had about half a dozen attacks of periodic paralysis. At the time Dr. Dana suggested that malaria might be at the bottom of his trouble. Some of the clinical aspects of the case simulated a low grade of poliomyelitis, but it was subsequently decided that the apparent atrophy of the muscles was the result of emaciation. The vasomotor symptoms predominated, and no sensory disturbances were present.

Dr. Taylor, in closing, said he thought the cases which had been mentioned by the various speakers were not absolutely analogous to those reported in his paper. In reply to a question, the speaker said that the sphincters in his case were not affected. In some of the cases the muscles of respiration were involved; in others not. In one case there was dangerous dyspnoea. No sensory changes could be found. The clinical aspects of the condition were so absolutely unique and characteristic that an error in diagnosis was hardly possible, viz., periodic paralysis of the muscles of the trunk and extremities, associated with loss of reflex and electrical excitability during the attack, and occurring as a family affection, with normal conditions between. The cases cited by the

various speakers evidently did not belong to this type, although possibly, they were related. Hysteria was evidently not to be considered, and malaria had no constant place in the etiology, nor was there sufficient evidence to show that the vasomotor system played any prominent part in its production. A temporary autointoxication seemed the most probable cause, the nature of which remains wholly unknown.

226. REMARKS ON SPINAL IRRITATION. Hugh T. Patrick, M.D.,
(*Medicine*, 3, 1897, p. 535).

The term "spinal irritation" should be banished from medical nomenclature. The pain and tenderness along the spine commonly known by this name have nothing to do with the spinal cord or its membranes, or with the spinal column. The condition is not due to congestion of anæmia of the cord, or altered nutrition, or a neurosis of the spinal arteries, or thickening of the membranes, or exhaustion of the gray matter of the cord, or an affection of the nerve roots or trunks, or irritation of vertebræ or spinal ganglia, or any other permanent condition in the back whatsoever. In so-called spinal irritation there are tender points along the spine. It can, however, be shown that these points shift rapidly, absolutely changing their position within five or ten minutes. The sore points are notoriously inconstant in degree of tenderness and in location, but it seems to be not generally known that the patient can locate them only about as accurately as a well person can locate a spot on his back previously touched. The tenderness cannot be due to anything abnormal at the tender spot, else the pressure the second time on the same spot would be just as painful as the first time, and, furthermore, we cannot suppose a pathological condition to have developed within five or ten minutes. This shifting corresponds just about to that in the healthy individual. If a normal person have any given spot on the back pressed, after five or ten minutes he will be unable to indicate exactly where the spot is. It seems reasonable to conclude that there is a lack of absolute accuracy of the sensorium. In the pathological case the shifting involves the sudden disappearance of pain from one place and its appearance in another. The inference is that the pain or pressure is itself due to the perverted mechanism of sensory reception and registration in the brain, or to a perverted reaction of still higher centres, constituting a vicious consciousness. A proclaimed diagnosis of spinal irritation may have an exceedingly bad effect upon a nervous patient. The presence of tenderness of the back with shifting sensitive spots, although indicating a functional nervous affection located entirely in the cerebrum, does not in any way preclude the presence, in addition, of organic disease of the brain, cord, or any other viscus. The almost instantaneous disappearance of tenderness from one point and its simultaneous appearance at another is not of itself proof of simulation, malingering, or nervous nonsense on the part of the patient.

FREEMAN.

Clinical Cases.

A CASE OF KATATONIC MELANCHOLIA.

By J. E. COURTNEY, M.D.,

First Assistant Physician of the Hudson River State Hospital, Poughkeepsie, N.Y.

The case described and illustrated is typical of that grade of melancholia which has been called "katatonic."



FIG. I.

The attack, which has lasted three years, was caused by a miscarriage. The patient is 38 years old. The trouble began with the symptoms of simple melancholia, depression, the delusion that she was in the way of her family, was ignored, hated and would be poisoned or killed in some way. She finally became violently agitated, and

her family could not care for her. When she came under the writer's care she was quiet and morose, stubborn and resistive, and kept her face covered with her hands; her only expression was: "I want to go home to Mamaronock." She had to be fed with a spoon, and was greatly irritated by all attentions. She soon settled into her present condition. She sits nearly all day with her face resting so firmly against her knees that impressions are



FIG. II.

made upon the temples from pressure. The arms are placed by the side of the legs, the hands resting in the popliteal spaces. This attitude is rigidly maintained for hours, and there is passive but rigid resistance to attempts to alter it. If the head is forcibly raised the eyes remain tightly closed, the head is turned slightly to the right and the cheeks are drawn in closely to the gums. If the patient is touched or spoken to or stimulated in any way, she says in a recitative manner the formula here il-

lustrated, the wording and tone of which are quite constant. Repetition of this can be induced any number of times by even slight stimuli. Beyond this she is absolutely mute. When called to meals, she gets up suddenly, rushes to the table with head lowered and little regard for intervening objects, eats rapidly with her fingers, and when excused resumes her usual pose. She sometimes voids urine in bed at night. There is considerable anæsthesia of the surface. She is much more manageable than

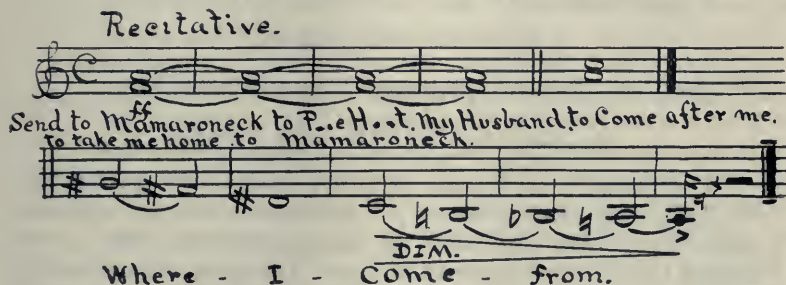


FIG. III.

when admitted, but otherwise her condition has not improved under treatment.

LES HEMATEMESES DES NEURASTHENIQUES (Hæmatemesis in Neurasthenic Subjects). (Journal des Conn. Méd., Dec. 3d, '96.)

Hemorrhages from the mucous membranes of neurasthenic subjects are not of rare occurrence, but hæmatemesis, though less frequent, has also been observed in quite a number of cases. Thus Mesnard, of Bordeaux, was the first to describe a case in a woman 52 years of age; M. F. Gallard reported two observations, and Dr. Ausset publishes a new case. This last observation presented a typical clinical picture of the disorder, the salient features of which were: Man, 37 years old, had first attack of hæmatemesis when 25, while preparing for an examination. Was subsequently well until March, 1896, when, after a violent emotion, he vomited blood again. The patient was in perfect health up to the time of the hæmatemesis, and on examination nothing abnormal was found in any part of his body, excepting a slight dilatation of the stomach. However, the patient was subject to severe cephalalgia, and was prostrated by the slightest exertion, besides being a confirmed neurasthenic. Dr. Gallard, having studied the symptoms of (specific) hæmatemesis in two neurasthenic subjects, considers it an easy matter to establish the diagnosis in such cases, the occurrence of vomiting blood in conjunction with the neuropathic symptoms, presenting a well-defined clinical entity.

MACALESTER.

Society Reports.

PHILADELPHIA NEUROLOGICAL SOCIETY.

April 25th, 1898.

The President, Dr. F. X Dercum, in the chair.

(Continued from page 622.)

Dr. F. X. Dercum presented the specimen from

A CASE OF CEREBELLAR TUMOR.

The patient was a man, 39 years of age, who presented the following clinical history. His family history was unimportant. He had had no diseases of moment, save scarlet fever at seven years of age. This was accompanied by destructive inflammation of the left middle ear, rupture of the drum membrane, and permanent impairment of hearing. Otorrhœa persisted for a year or more, and subsequently ceased. When he was about 28 or 29 he contracted syphilis, for which he was very thoroughly treated. Some six years ago he began to suffer from pains referred to the left brow and left temple. This pain was at first regarded and treated as a neuralgia. It was quite persistent for about a year. Occasionally exacerbations of the pain occurred at night, and at times the pain was referred to the teeth. Subsequently the pain became less marked, but never entirely disappeared, and in 1893 it recurred with full force. It was referred at this time to the back of the eye, and was made much worse by jars and sudden movements of the head. He suffered occasionally also from double vision, but this symptom subsequently disappeared. The pain in the head subsided somewhat, and, with the exception of occasional recurrences in the left temple, he presented few symptoms, save that he was very much indisposed to exertion, either physical or mental. He noticed in the summer of 1896 that he began to be unsteady when riding his bicycle, and later on unsteadiness was also noticed in walking.

When he was first seen by Dr. Dercum, in the spring of 1897, the sway was decidedly plus, and the gait quite ataxic, and at times titubating. No local palsies and no ataxia of the hands or arms were present. The knee-jerks were plus, but the legs were flaccid. No sensory disturbances and no rectal or vesical symptoms were noted. Tremor also was absent. The tongue was protruded in the median line. The pupils were equal and responsive to light. The movements of the globes appeared to be normal. An examination of the eyegrounds revealed a beginning optic neuritis. Speech was at this time somewhat slow; at times drawling. Little change was noted in the case from time to time, save that during the following summer he became decidedly more ataxic, while a slight weakness of the left arm and leg also made its appearance. By November the optic neuritis had become quite pronounced. Headache was again quite severe, and about this time he began to suffer from epileptiform seizures, general in character and exceedingly brief in duration. They generally occurred during the night. When occurring during the day they most frequently assumed the form of a slight petit mal. Subsequently the optic neuritis attained a very high grade, and in January of 1898 beginning loss of vision was noted. The tongue now deviated slightly to the right. The left side of the face and the left eyelid drooped slightly. The paresis of the left arm and leg also became a little more marked, and speech more distinctly drawling in character. Loss of vision gradually became more pronounced, and epileptiform seizures more frequent. The patient subsequently was bedridden, and progressive mental failure, coma, and death finally ended the scene.

The autopsy revealed a large, caseous, friable tumor, involving the base of the left lateral lobe of the cerebellum anteriorly. The adjacent portions of the pons and medulla oblongata had evidently been somewhat pressed upon, and to this fact the cranial nerve symptoms present, namely, the deviation of the tongue and the slight facial palsy, are to be ascribed. The growth appeared to be tuberculous, and special interest is derived from the fact that the adjacent portion of the temporal bone was necrosed. The query naturally suggests itself whether this large tuberculous deposit may not have had its origin in

tuberculous necrosis of the temporal bone, the latter itself being a result of the chronic otitis media.

Dr. James Hendrie Lloyd exhibited

A TUMOR OF THE CEREBELLUM.

The patient had been entirely blind and deaf, and had suffered from intense headache and occasional vomiting. He had been unable to stand. No involvement of the cranial nerves had been noticed. There was a tendency to pitch forward. At the necropsy a tumor, fully as large as a hen's egg, was found in the superior part of the vermis. The corpora quadrigemina had been destroyed by pressure. The attachments of the tumor were slight, and it could easily have been removed.

Dr. Judson Daland reported

A CASE OF MENINGOMYELITIS.

The patient was a female, aged 19. The present illness began June 1st, 1896. Several days previous to this the patient went to the cemetery, and was on the damp ground for some time. The day before she became very much overheated from dancing, and was exposed to a draft.

On rising on the morning of June 1st she experienced considerable pain in the back, about the last lumbar vertebra, which she compared to a toothache. This remained localized, but was not severe enough to prevent her being up and about all day until evening, when it became very severe. She was unable to sleep that night on account of the pain.

The next day she arose with a very severe headache, which continued throughout the night. She had considerable fever, varying between 100 and 105 degrees F., but was able to walk about all the day. The lumbar pain was somewhat less, but the headache continued. She did not sleep that night on account of aching in the lower extremities. The following morning, the third day, at 1 A. M., she attempted to get out of bed, but fell and was unable to rise. She could raise the left leg with great effort, but the paralysis of the right leg was complete. The bladder and rectum were also paralyzed. Pain and fever continued to be constant symptoms, but the head-

ache disappeared. The night of June 3d, the lumbar pain was considerably decreased, and the pain disappeared from the legs. Sleep was secured by the use of opiates. During the following two days, Thursday and Friday, her condition remained unchanged. Friday evening the paralysis became complete in the left leg. She noticed that she had lost all power to use the left arm, that the fingers were flexed and the hand flexed on the forearm, but she could move the fingers and toes. The reflexes were not noted. The fever disappeared, and on the fifth day a marked tendency to drawing backward of the head was noted. Lifting the head or turning it to either side caused pain. She was quite excitable. Her sight and hearing were not affected. Although the pain had disappeared, the hyperaesthesia was marked.

Her condition remained unchanged for 90 days, when improvement began. She became able to sit up in bed, and later in a chair, and to move the left leg sufficiently to rock the chair in which she was sitting. The improvement in the arms continued, and, to a slight extent, in the legs. Her general health was unusually good; the vital functions of the body being normally performed, with the exception of the bowels, which were obstinately constipated. Several analyses of the urine showed the secretion to be normal.

The muscles of the neck, head and upper extremities are at present well developed, and well covered with adipose, except those of the left forearm, arm and shoulder region. The left deltoid and supraspinous muscles are considerably atrophied. The left biceps and triceps, although smaller than the right, are in good condition. The muscles of the left forearm are smaller than those of the right. The thenar and hypothenar groups of muscles of the left hand are almost completely atrophied, and the entire hand seems smaller. The grip of the left hand is diminished in force about one half. The muscles of the right arm are unusually strong. The left arm is strong also, but is weaker than the right. The left arm is brought to a right angle with the body with difficulty, and the movement is with pronation. It is exceedingly difficult to raise the left arm above this point, and it is impossible to carry it backward beyond the midline of the body. The lower extremities are in extension. The feet can be slightly

moved. Resting on her right side, with leg partially flexed, she flexes and extends the left leg on the thigh well, but these movements are overcome by a weight of a few ounces. The same is true of the right leg, to some extent, but motion is considerably more restricted than in the left leg, and it is also necessary to support the weight of the leg by the hand. When lying on her face she can raise her left foot about an inch from the bed. When the leg is flexed at the knee she can hold it fixed at a right angle with the thigh. On examination the spine shows considerable left lateral curvature, which extends about an inch and a quarter from the perpendicular.

Dr. Daland spoke of the large daily amount of iodide of ammonium (6 1-3 drachms) and of bichloride of mercury ($\frac{3}{4}$ grain) the patient had been taking.

DISCUSSION.

Dr. Wharton Sinkler did not believe that any special significance regarding the nature of a disease could be drawn from the amount of iodide a person is able to take. Some syphilitic persons will tolerate only a small amount, while others, not syphilitic, can take the drug in large doses.

Dr. F. Savary Pearce reported

A CASE OF ATAXIC PARAPLEGIA, WITH SEVERE PAIN AND MUSCULAR SPASMS, FOLLOWED BY BLOOD EXTRAVASATIONS.

Reference was made to the rarity of ecchymoses following muscular spasms, and to the paper by Weir Mitchell,¹ in 1869, in which five cases were reported.

J. I. T., male, an American, 33 years of age, of a gouty ancestry, had always been a healthy, active ranchman, living at an altitude of 6,000 feet for the past nine years. He suffered from gravel at so early an age as five years. This is mentioned on account of the uric acid heredity. Seven years ago he had a small chancre, was insufficiently treated for four months, and, considering himself well, continued actively at ranching in the apparent vigor that is so common in those living at such an altitude. Six years ago, while again on his annual visit to the coast line, he began

¹Transactions of the College of Physicians of Philadelphia, vol. iv., N. S., pp. 282-288.

to suffer from "catching cold" easily, and one day he was suddenly taken with anorexia, headache and chill, which were soon followed by unconsciousness, without convulsions. He remained generally paretic, with flaccidity of the muscles, and continued delirious for about a fortnight. He gradually recovered, after a month, sufficiently to be about; but convalescence was unsatisfactory. After another month he returned to the mountains. Four years ago he descended to the coast a second time, and had a renewal of his spinal meningeal symptoms. He soon began to suffer from numbness and increasing stab-like pains in the thighs and calves. All four extremities became stiff, and he was somewhat relieved by moving about or stretching himself. Recurrences of pain-spells in the calves of the legs gradually became unbearable. He became decidedly spastic and ataxic. Two years ago he began to have firm contractions of the thigh and calf muscles, with the attacks of algæsia described as atrocious, and occurring mostly in the early morning or in the evening.

After the severest attacks of pain he has found "black and blue marks" at the site of the most painful areas, and distal to them, not resulting from any pressure on the parts. This would exclude trauma as the etiological factor. The extravasations were not seen until some three to five hours after the painful seizures, and were rather deep, and disappeared slowly, as after a bruise. They occurred only half a dozen times, and at the height of the pain. The extravasations were from one half to one inch in circumference, and with diminishing pain the tendency to extravasular leakage also ceased. One year ago the bladder became involved.

On examination he presented the typical syndrome of marked ataxic-spastic paraplegia. The arms were somewhat involved. Argyll Robertson symptom was not noted, although in the past a transient diplopia had existed. Romberg's symptom was marked. The urine showed a trace of albumin from pus, but no indican or uric acid excess and no glycosuria. An important feature of the case was the very great improvement in the ataxia, the spasticity and the general health of the patient, brought about by the combination of "rest" treatment with head extension, followed by potassium iodide up to gr. cccl. daily, and later coördinated voluntary movement of arms and legs.

DISCUSSION.

Dr. John K. Mitchell said he had instructed this patient for some time in graduated movements. From being so ataxic that he could not stand, he became able to walk across the room, putting one foot directly in front of the other, and to walk backward in the same way, which is not an easy feat for even a normal individual.

Dr. Spiller reported a

CASE OF MERALGIA PARÆSTHETICA.

A man of middle age had disturbance of sensation, closely confined to the distribution of the external cutaneous nerve of the right thigh. When in bed or sitting in a chair, or immediately after standing upon his feet, no unpleasant symptom was noted; but after standing for a short time, or walking, a distinct burning and drawing sensation, attended with severe pain, was experienced in the distribution of the nerve named. Sensation otherwise and the condition of the muscles were normal. The affection had existed for one year, but had become more marked during the past few months. Dr. Spiller thought that if other means failed to produce relief, stretching, or later even cutting the external cutaneous nerve, might be advisable, inasmuch as no muscular paralysis would result, and the nerve could be easily reached at the anterior superior spine of the ilium, and, at the most, loss of sensation in a limited portion of the thigh would probably be the only unpleasant result. He preferred the name of meralgia paræsthetica to the rather cumbersome one of Bernhardt's disturbance of sensation in the thigh.

DISCUSSION.

Dr. Wharton Sinkler had seen two cases of the disease described by Dr. Spiller. One patient, a woman, had had the symptoms in the distribution of the external cutaneous nerve referred to. After several months she recovered. The second case, now under observation, was in a patient who suffered injury in a railroad collision. Intense burning pain was experienced in the distribution described.

Dr. F. Savary Pearce had seen the same condition in a case where the middle cutaneous nerve of the thigh appeared to be alone involved. This was a gynæcological case. The patient had had sepsis, and had been subjected to operation upon the bladder and womb, and it was a question whether the irritation had not extended from this source.

Periscope.

With the Assistance of the Following Collaborators:

CHAS. LEWIS ALLEN, M.D., Wash., D.C. R. K. MACALESTER, M.D., N.Y.
 J. S. CHRISTISON, M.D., Chicago, Ill. J. K. MITCHELL, M.D., Phila., Pa.
 A. FREEMAN, M.D., New York. H. PATRICK, M.D., Chicago, Ill.
 S. E. JELLIFFE, M.D., New York. JOSEPH SAILER, M.D., Phila., Pa.
 WM. C. KRAUSS, M.D., Buffalo, N.Y. HENRY L. SHIVELY, M.D., N.Y.
 W. M. LESZYNSKY, M.D., New York. A. STERNE, M.D., Indianapolis.

ANATOMY AND PHYSIOLOGY.

227. ZUR HÄRTUNG DES CENTRALNERVENSYSTEMS IN SITU (On the Hardening of the Central Nervous System in Situ). Pfister (Neurologisches Centralblatt, 17, 1898, p. 643).

The author uses a five to ten per cent. solution of formaldehyde and injects it directly into the vertebral canal through the third and fourth intervertebral spaces. For the brain a like procedure through the superior orbital fissure is adopted. JELLIFFE.

228. CONTRIBUTION A L'ÉTUDE DES LOCALISATIONS DES NOYAUX MOTEURS DANS LA MOELLE ÉPINIÈRE (Contributions to the Study of the Localization of the Motor Nerves in the Spinal Cord). G. Marinesco (Revue Neurologique, 6, 1898, p. 463).

Marinesco, by complete or partial destruction of the motor nerves in cats and dogs and guinea pigs, presents the results of his studies in the following brief résumé:

(1) The cutting off of a spinal nerve from the spinal axis produces a condition of chromatolysis which is characteristic.

(2) Such a reaction can be utilized in order to determine the topographical position of the nucleus of origin of a motor nerve or set of nerves.

(3) As a rule each motor nerve arises from a main nucleus and an accessory nucleus, the main nucleus being plainly localized in the majority of cases. The median and cubital nerves prove exceptions to this general rule.

(4) Each motor spinal nerve arises from more than one medullary segment; two, three or even more nerve segments may contribute to its formation. JELLIFFE.

229. ON THE STRUCTURAL ALTERATIONS OBSERVED IN NERVE CELLS. W. B. Warrington (Journal of Physiology, 23, 1898, p. 112).

Using Held's method of staining the author experimented with cats and rabbits and comes to the following general summary in this excellent paper.

(1) Distinct and easily recognizable changes in nearly all the cells of a segment of the spinal cord are found on the side of the lesion after section of an anterior root.

(2) Similar but less marked changes follow division of the facial nerve, and still less distinct alteration after division of the oculomotorius nerve.

(3) The fate of such altered cells and the ultimate condition of the nucleus of origin are not yet definitely ascertained.

(4) The age and nature of the animal experimented on is a factor in determining the rapidity and degree of alteration met with in nerve cells.

JELLIFFE.

PATHOLOGY.

230. ZUR LEHRE VON DER SYRINGOMYELIE (On Syringomyelia). L. Minor (Zeitsch. f. Klin. Med. 34, 1898, p. 373).

The author presents a study of two cases of syringomyelia. The first case occurred in a one-year-old child, who from birth presented a number of anomalies of the nervous canal. It had spina bifida, meningocele and hydrocephalus. Microscopic examination showed hydromyelia, syringomyelia with duplication of the central canal with well marked continuity of the two cavities. In the second case similar teratological developments were noted. This second case was in a twelve-year-old girl, who died of a compression myelitis due to an endothelioma. Microscopically there was found in the upper segments of the dorsal cord a marked dilatation of the central canal. In this case the syringomyelia could be demonstrated, by a study of the serial sections, to have originated from a cut-off portion of the central canal. The author, as a result of his studies on these two cases, emphasizes the Leyden embryological view as to the origin of syringomyelia.

JELLIFFE.

231. SULLE LESIONI DEL SISTEMA NERVOSA CENTRALE PRODOTTE DAL BACILLO ICTEROIDE. Dott. A. Cesaris-Demel, Torino (Giornale della R. accademia di medicina di Torino, Marzo, 1898).

In the pathological laboratory of Professor Foa, of Turin, Dr. Cesaris-Demel experimented on the action of the bacillus of yellow fever as described by Sanarelli, on the central nervous system of dogs and rabbits, and came to the following conclusions:

(1) The icteroid bacillus produces in animals (dog and rabbit) important lesions in the cells of the central nervous system.

(2) These lesions affect principally: (a), the large and middle pyramidal cells of the cortex, which become swollen, lose their coloration and continuity of the chromophilic elements, arriving at a complete destruction of the cell; (b), the large cells of Purkinje which lose the coloration of the prolongations, and present a swelling of the basal part with intense chromatolysis and destruction of the cellular membrane; (c), the pyramidal cells of the anterior horns and of the oblongata, which present a conspicuous chromatolysis of a peripheral type.

(3) These lesions stand in relation to the gravity and extension and more or less susceptibility of the animal to the icteroid bacillus and the infection.

KRAUSS.

232. ZUR PATHOLOGIE DER EPILEPSIE (The Pathology of Epilepsy). N. Krainsky (Allg. Zeitsch. f. Psychiatrie, 54, 1897, p. 612).

From an extended series of examinations of the blood and urine the author comes to the general conclusion that the epileptic poison

is to be found in the blood. The chemical nature of this poison is similar to carbamic acid, and a detailed report is given of a series of experimental researches. JELLIFFE.

CLINICAL NEUROLOGY.

233. EIN WEITERER FALL VON SOLITÄRER TUBERCULOSE DES RÜCKENMARKS, ZUGLEICH EIN BEITRAG ZUR LEHRE VON DER BROWN-SÉQUARD'SCHEN HALBSEITENLÄHMUNG. (Another Case of Solitary Tuberculosis of the Spinal Cord, at the Same Time a Contribution to the Brown-Séquard Paralysis). L. R. Müller (Deutsche Zeitschrift für Nervenheilkunde, vol. 12, 1898, p. 288).

A man, 46 years old, who had phthisis, became very weak in the right lower limb, while the power in the left was retained. He had retention of urine, exaggerated knee-jerk, especially on the right side, and disturbed sensation on the left side. The upper extremities were not affected. The senses of pain and temperature were completely lost on the left side below the ribs. The senses of touch, pressure and location were not disturbed anywhere. Sensation was somewhat impaired in a small area over the right nipple. A solitary tubercle was found in the right side of the second thoracic segment of the spinal cord. The left half of the cord was compressed by the growth, but otherwise very little altered. Although the clinical signs of spinal disease had lasted five weeks, secondary degeneration was not very important. A slight degeneration was noted in the left anterolateral column, beginning a little above the tubercle. This, the writer believes, was due to destruction of the gray matter, especially of the posterior horn, on the right side. It is only in rare cases that the existence of this tract in the anterolateral column, arising in the contralateral gray matter, has been indicated by pathological findings. The vertebræ and membranes were normal. A slight degeneration of the posterior columns in the lower thoracic and lumbar regions was supposed to be similar to that seen in pernicious anæmia and marasmus. The diagnosis of a solitary intramedullary tumor in the right half of the cord was made before death, and was founded on clinical phenomena, in a phthisical patient, indicating a partial transverse lesion. The free movement of the vertebral column, the absence of painful areas along the spine, the painless development of the hemiplegia, were regarded as indicative of the integrity of the spinal vertebræ and membranes. Müller agrees with Bruns that in spinal paraplegia the lesion is usually located too low in the cord.

Although only one-half of the cord was destroyed, the paralysis was not truly of the Brown-Séquard type. The weakness of the right lower limb did not amount to paralysis, and the disturbance of sensation on the left side was syringomyelic in type. The existence of naked axis cylinders, which escaped detection within the tubercle, may possibly explain the partial paralysis, but not the disturbed sensation. The views concerning Brown-Séquard paralysis are correct as regards pain and temperature senses, but not as to tactile sense; the former senses, the author thinks, are altered by lesion of the posterior horn of the side on which the fibres enter the cord, and by lesions of the anterolateral column of the opposite side.

SPILLER.

234. SUR UN CAS DE MYÉLITE SUBAIGUE DORSO-LOMBAIRE [Sub-Acute Dorso-lumbar Myelitis (due probably to infection by way of the uterus)]. Mongorier et Carrière (La Presse Médicale, Vol. 55, 1897, p. 8).

The authors describe a case of myelitis affecting the portion of the cord below the seventh dorsal segment, and give a very complete

account of the changes found both in the cells and fibre tracts and in the vessels of the cord on examination by recent staining methods. They suggest that the disease may have been the result of an infection from the uterus, as the patient had had for some time an unhealthy condition of the endometrium, and had been subjected to the operation of curettage about a month before the onset of the myelitis.

ALLEN.

235. PATHOGÉNIE DE LA RIGIDITÉ MUSCULAIRE ET DE LA CONTRACTURE DANS LES AFFECTIONS ORGANIQUES DU SYSTÈME NERVEUX (Pathogenesis of Muscular Rigidity and Contractures in Organic Disorders of the Nervous System). A. Van Gehuchten (La France Méd., 44, 1897, p. 629).

The physiological mechanism of muscular rigidity and contraction, which are constant symptoms in organic hemiplegia and spasmodic paraplegia, are discussed by the author. The theories advocated by Follin, Hitzig, Straus, Marie, Freud, Jackson and others, according to which these phenomena are of muscular, spinal or cerebellar origin, prove to be inadequate in explaining the clinical and anatomical facts. Besides, it would be difficult to find a solution applicable to both hemiplegic and spasmodic contractures, the underlying cause not necessarily being the same. The author maintains that spasmodic contraction is an active process, that is, an exaggeration of normal muscular tonus, of *cerebral* origin, due to interruption in the course of the cortico-spinal fibres, with preservation of cortico-ponto-cerebello-spinal nerves, which latter keep the cells of the spinal cord under the control of the motor cells of the cortex. On the other hand, hemiplegic contracture has quite a different genesis, the influence of the cerebral cortex upon the motor spinal cells and the corresponding paralysed muscles being cut off by the lesions, and if contractures develop in this case, they are of peripheral origin, due to a difference in degree of paralysis between the flexor and extensor muscles. The former are usually less involved than the latter, therefore the frequency of post-hemiplegic flexion contractures. If, on the other hand, the paralysis is of equal intensity in all the muscles of the extremity, no contractions will result, and there will be a flaccid condition.

MACALESTER.

236. DES PARALYSIES POST-ANESTHÉSIQUES (Post-Anæsthetic Paralysis). Ed. Schwartz (Gazette des Hôpitaux, 70, 1897, p. 1,248).

Following operations performed under general anæsthesia there are sometimes observed paralyses which have no pathological connection with the seat of operation, and surprise both patient and surgeon by their unexpected appearance. The following belongs to this class of cases:

A man, æt. 45, was operated on for a small inguinal hernia. During the operation, which was performed under chloroform anæsthesia, there was a slight attack of cardiac and respiratory syncope, which was easily and promptly overcome by artificial respiration. On emerging from the chloroform narcosis the patient observed a feeling of formication in the right hand, especially in the thumb and index finger, and he could not readily move the two affected fingers. The next day there was a complete paralysis of the flexor longus pollicis and the flexors of the index finger. All the other muscles of the upper extremity were intact. There was no anæsthesia or hyperæsthesia. When he tried to move his lower limbs there was observed a well-defined paralysis of the right quadriceps extensor femoris. The paralysis was thus limited to the right side, involving

a single muscle below and two above. These symptoms gradually disappeared under massage and electricity, and at the expiration of seven months there was complete cessation of the paralysis.

When the paralysis of the thumb and index finger was observed the first impression was that the case was one of peripheral paralysis due to stretching of the brachial plexus, and yet there had been no violence used in applying artificial respiration, which had lasted barely two minutes. Moreover, it was no longer possible to consider this origin of the trouble after the development of the quadriceps extensor paralysis, which could only be assigned to a central origin, functional or otherwise. Upon carefully questioning the patient, it was learned that for some time he had noticed a little clumsiness in using his right hand, and that in writing it had become necessary for him to use a thicker penholder. This fact strengthened the assumption of a central paralysis. The possibility of hysteria in this case could be ignored.

Post-anæsthetic paralyses, as they are usually classified, show two distinct types. Sometimes they are true peripheral paralyses, especially of the brachial plexus and its branches, occurring thus more commonly in the upper extremity. These cases have been observed by many surgeons, and present the clinical features described by Erb, involving usually the deltoid, brachialis anticus, biceps and supinator longus. More rarely other muscles are paralyzed. These cases are true traumatic paralyses, caused by compression or stretching in long-continued or strained faulty positions, or to the circualar compression of limbs by tight elastic bands. They may, and should, be avoided by carefully watching the patient during the administration of chloroform or ether. This first class of cases then is not true postanæsthetic paralysis, if this term is intended to have any etiological significance, for they have no other relation to the anæsthesia than to appear consecutive to it, and to have been produced by a vicious attitude during the continuance of the narcosis.

Much more rare and much less studied is another class of cases, which cannot be explained in the same manner, and which presents a different clinical picture. Without being committed to any particular theory as to their nature, they may be called, as the writers who have studied them have done, true postanæsthetic or central paralyses. The case here reported is such a one, and there have been collected in all ten cases, the clinical appearances of which are variable, but which may be arranged in three classes:

1. Cases in which the paralysis is limited to a single limb, monoplegias resembling the cases described by Büdinger and Franke, the first, with autopsy, showing a zone of softening in the cerebral cortex, the second occurring in a hysterical woman with the symptoms of hysterical paralysis.
2. Cases in which are observed paralyses of a cranial nerve (the facial) alone or associated with paralysis of the limbs or of other cranial nerves.
3. Cases in which true hemiplegia occurs, either observed immediately upon emerging from the sleep of anæsthesia, or developing a short time after.

The pathological question which is presented is difficult of solution. Is there here a toxic paralysis, as believe Büdinger and Burnheim, comparable to alcoholic paralyses, and depending on a special action of chloroform, causing a degeneration of nerve tissue when the latter constitutes a *locus minoris resistantiæ*? Such would not seem to be the case owing to the rarity of its occurrence, and to the fact that it is observed in cases where the anæsthesia has been of very

short duration or incomplete. May it be accounted for as a simple coincidence? It is possible, but not demonstrable. On the other hand, it may well be believed that where endarteritis exists that patients with diseased arteries may during the period of excitement, while crying out, struggling or vomiting, rupture a vessel and present variable symptoms, depending upon the site and extent of the lesion. The cases of Depoge, Gross and Büdinger harmonize with this view. Finally, hysteria may account for a certain number of these cases, and in the present day it may be well to explore this field further.

From the practical standpoint it is impossible to foresee its occurrence, but it may be suspected where arterial or cardiac disease is present. Recovery often takes place when there is not total hemiplegia. SHIVELY.

237. VON DER VERWACHSUNG ODER STEIFIGKEIT DER WIRBELSÄULE (Concerning Ankylosis or Rigidity of the Spinal Column). W. v. Bechterew (Deutsche Zeitschrift für Nervenheilkunde, 11, 1897, p. 327).

In a previous communication v. Bechterew described an affection consisting of (1) immobility or rigidity of a part or the whole of the spinal column, without distinct tenderness on percussion or on bending the body; (2) of kyphosis, especially in the upper thoracic region, causing the head to project forward; (3) of paresis of the muscles of the trunk, neck and extremities, with unimportant atrophy of the muscles of the back and scapulæ; (4) of diminished sensation, especially in the areas of the cutaneous branches of the thoracic, lower cervical, and sometimes of the lumbar nerves; (5) of irritation symptoms (paræsthesia, pain) in the distribution of these nerves; (6) occasionally of spasmodic twitching or contracture (irritation of motor nerves) of the muscles in the extremities; (7) of reaction of degeneration in advanced cases.

On account of the paresis of the muscles the erect position of the head is rendered difficult. The breathing is abdominal. Heredity and trauma seem to play a rôle, at times, in the development of the disease. The affection is progressive, and is little known. The "arthritis deformans of the vertebral column," described by Oppenheim, does not correspond in every respect with the rigidity of the vertebral column described by v. Bechterew. A new case of this uncommon affection is described at length.

As yet no necropsies have been obtained, but it is probable that the vertebræ become united, that the spinal nerves are thereby compressed, and that the inflammation extends to the spinal dura.

SPILLER.

238. BEMERKUNG ÜBER DIE CHRONISCHE ANKYLOSIRENDE ENTZÜNDUNG DER WIRBELSÄULE UND DER HÜFTGELENKE (Remarks on the Chronic Inflammation of the Vertebral Column and Hip Joints). A. Strümpell (Deutsche Zeitschrift für Nervenheilkunde, 11, 1897, p. 338).

The above paper by v. Bechterew has called forth the statement from Strümpell that he has been acquainted for a long time with this peculiar form of chronic arthritis, which affects the vertebral column and hip joints, and causes complete ankylosis of these parts. He publishes the third case of this peculiar disease observed by him. He is somewhat in doubt as to whether the disease which he describes is exactly the same as v. Bechterew's affection or Oppenheim's "arthritis deformans of the vertebral column." SPILLER.

239. LECTURES ON APHASIA. Bramwell (Edinburgh Medical Journal, July to December, 1897).

These lectures are an elaboration of those given by the author in his course on medicine. He makes the usual divisions of aphasia into (a) word deafness, (b) word blindness, (c) motor vocal aphasia (aphemia), (d) motor writing aphasia (agraphia), and proceeds to describe the chief characteristics of each variety. The close connection between the centre of auditory word memories and the speech centre and that between the centre for visual word memories and the writing centre is emphasized. Speaking of word deafness, he suggests that the character of the lesion, whether irritative or destructive, is of importance, as where it is only irritative there may be paraphagia or paraphasia, instead of complete loss of power to write and to speak. When the centre for visual word memories is destroyed, there may be still ability to write through the revival of memories of movements presented in the kinæsthetic centre for writing movements, but this power can hardly extend further than to the production of syllables and short words, not to complete sentences. It is certain that destruction of the visual speech centre does not produce paralysis of the movement of the hand and arm, but it is a question whether or not, under the circumstances, the finer movements, such as piano playing, etc., do not suffer to some extent, and observations on this point are needed.

The author is of the opinion that a separate writing speech centre does not exist, but that the centre for writing movements is included in the psychomotor centre for movements of the hand and arm. Whether the kinæsthetic memories of the movements made use of in writing are stored up in the same region from which the motor impulses emanate, or elsewhere, is uncertain. Speaking of other varieties of sensory aphasia, it is suggested that in the blind, who read by sense of touch, as the result of a lesion "tactile aphasia" may be produced, and it is urged that physicians to blind asylums investigate the matter.

The aphasic symptoms due to interruption of the connecting and commissural fibres passing between the different speech centres are next considered, and after this follows a discussion as to the relative activity of the corresponding speech centres in the two hemispheres of the brain. While one side of the brain (the left in right-handed people) contains the leading or "driving" centres, the action of the centres upon the opposite side should not be underestimated, as it is doubtless important. In support of this point, a number of facts are mentioned, and interesting suggestions are made. The question of blood supply to the different centres is taken up, and it is pointed out that most of them are situated about the boundary lines of regions supplied by different centres, and as these boundaries vary somewhat in different brains, we may have here an explanation of the different symptoms resulting from the plugging of a certain artery in different cases. Lastly follows a discussion of the physiology of speaking and writing, and the development and education of the different centres concerned in these processes. This is one of the most interesting and instructive parts of the course. The lectures are clearly and pleasantly written, and give an excellent résumé of the subject. ALLEN.

240. ZUR LEHRE VON DER GLEICHSEITIGEN HEMIPLEGIE BEI CEREBRALEN ERKRANKUNGEN (A Contribution to the Study of Collateral Hemiplegia in Cerebral Diseases). N. Ortnier (Deutsche med. Wochenschrift, 23, 1897, p. 372).

Ortnier mentions a valuable diagnostic sign in cases of collateral hemiplegia, i. e., those in which the paralysis is on the same side

as the cerebral lesion. He reports two cases of left-sided hemiplegia, in each of which he correctly made the diagnosis of a left-sided cerebral lesion, because the degree of the respiratory movements of the right side of the thorax was much below normal, while on the left side the movements were not affected. He acknowledges that in rare cases of contralateral cerebral hemiplegia differences in the respiratory movements of the two sides of the thorax are not found. It is not the condition of the extremities, but that of the respiratory muscles which is of decisive moment for the localization of the lesion in cerebral hemiplegia.

After mentioning and rejecting the various theories advanced to explain hemiplegia existing on the side of the cerebral lesion, Ortnér accepts the views of Pinneles. There is not a true paralysis of the limbs on the same side as the lesion in these cases, but a relaxation of the muscles, and, in the limbs of the side opposite to the lesion there are manifestations of cerebral irritation, such as apparently voluntary movements of defense. As such symptoms of irritation are absent in the limbs on the same side as the lesion, the appearance of paralysis is produced. In none of thirty-eight cases of collateral hemiplegia, with autopsies, taken from the literature was there any statement of a lesion of the internal capsule, and in all these cases the lesion, from its location, was well adapted to cause signs of irritation in the opposite half of the body.

Collateral hemiplegia is comparatively frequent in hæmatoma of the meninges or in internal hemorrhagic pachymeningitis, and the diagnosis of these conditions becomes possible in cases in which apparent paralysis of the limbs, and, possibly, also of the face, of one side offers a striking contrast to the normal movements of the respiratory muscles of the same side. Trephining in such cases should be done on the side on which the hemiplegia is noted.

The observation of imperfect respiratory movements on one side will lead to a correct diagnosis of the side of the brain affected in cases in which bilateral symptoms of irritation or flaccid paralysis of the extremities are present.

SPILLER.

PSYCHOLOGY AND PSYCHIATRY.

241. A STUDY IN APPERCEPTION. W. B. Pillsbury (American Jour. of Psychology, 8, 1897, p. 315).

The author presents Wundt's views. In normal consciousness, at any time, some ideas will be found to be prominent and distinct, while others are vague and indefinite, gradually shading down to obscurity from "the point of clearest vision." Clearness of ideas is not the same as intensity of sensation, but intensity favors clearness, and clearness favors intensity. Degree of clearness varies inversely with the number of ideas simultaneously contained in the point of clearest mental vision. Preceding, accompanying and succeeding increase in the clearness of ideas there is a varying complex of sensational and affective phenomena, viz., (1) increase of clearness in the idea directly before the mind, accompanied by the immediate feeling of activity; (2) inhibition of other ideas; (3) muscular strain sensations with the feelings connected with them, intensifying the primary feeling of activity; (4) the reflex of these strain sensations intensifying the idea apperceived. An idea never undergoes the peculiar increase in clearness when these phenomena are not present. The change of clearness is not like quality or extent and other attributes of sensation. It takes place while all external conditions remain the same. The whole circle of subjective processes connected with apperception Wundt calls "attention".

Passive apperception is present (1) at certain times when an idea

enters consciousness under the most favorable conditions, and (2) it sometimes precedes active perception. It is never so complete or full as active apperception, and in the typical form it is determined immediately and without choice. In active apperception the incentives are more numerous and more evenly balanced, and the decision regarding the merits of ideas is equivocal, and often delayed. Apperception of any sensation is rendered easy by its separate and isolated appearance shortly before in consciousness. No sensation comes to its full rights in consciousness unless apperceived. CHRISTISON.

242. INVOLUNTARY MOTOR REACTION TO PLEASANT AND UNPLEASANT STIMULI. G. V. Dearborn and F. N. Spindler (Psychological Review, 4, 1897, p. 453).

The authors experimented with reference to the hypothesis of Prof. Munsterberg, that stimuli, which cause action of the extension muscles, are as a rule, agreeable, while stimuli which cause action of the flexor muscles are, as a rule, disagreeable. According to this theory, the hands should relax and the head drop back under agreeable stimuli, while under disagreeable stimuli, the reverse should take place.

The hands and the head were, therefore, chosen as the reacting organs of the experiments. The stimuli used were odors, sounds and colored light. It was much more difficult to find for each subject a positively disagreeable odor, than it was to find a positively pleasant one. The most emphatic were: bergamot, cologne water, heliotrope, methyl acetate, oil of cloves, tincture of musk, ethyl iodide, spirit of turpentine, xylol, eugenol, oil of eucalyptus, iodoform, cider vinegar, bisulphide of carbon, ethyl bromol and camphor, sulphuric ether, toluidin, allyl alcohol, tincture of asafetida, diamylamine, acetic acid, ammonium valerianate.

It was expected that as the lower animals, savages and children are very responsive to sensory stimuli, the effects of civilization or education would reduce motor manifestations as responses to sensory stimuli. It was found, however, that some subjects did not react at all, except to pronounce the stimulus pleasant or unpleasant, and other subjects would give a motor reaction while they pronounced the stimuli indifferent. Others, again, were so sensitive that "they seemed to go all to pieces" by any disagreeable stimulus, and would "show most surprising and seemingly contradictory reactions."

The subjects cover mostly seniors and juniors of the Harvard and Radcliff colleges and graduates working in the laboratory. They were nineteen in all. They were each comfortably seated in an arm-chair, and their heads and hands ingeniously connected with registers. The summary is as follows. It includes only actual reactions to stimuli, 764 reactions in all. The cases where stimuli were applied without resulting reactions numbered 253.

	Under Unpleasant Stimuli.	Under Pleasant Stimuli.	Under Indifferent Stimuli.
Flexion.	66.6	32.2	49
Extension	33.3	67.8	51
Proportion	2 to 1	1 to 2 +	Nearly equal.

These experiments afford a striking confirmation of Professor Munsterberg's theory that there is a strong tendency to expansion

under agreeable and contraction under disagreeable stimuli. Other tendencies are present, however, some of which conflict with this one, such, for example, as the tendency to move toward an object which attracts attention; the tendency to move away from a disagreeable object; the tendency to make particular movements of adaptation to stimuli, etc. A further influence of great interest is revealed upon examination of the records of the separate individuals who as subjects took part in these experiments. If their reaction to stimuli what they pronounced indifferent be examined, it will be seen that some show a temperamental tendency to make movements of flexion more often than of extension; others, the opposite, and others still to make both in nearly equal proportion.

The "flexion" temperament shows through the greater predominance of flexions a greater difference in the proportion of the two movements under pleasant stimuli and a nearer approach to equality under unpleasant stimuli. The "extension" temperament shows the opposite results, and the indifferent temperament exhibits proportions more nearly those given in the above table.

Temperamental differences then work together with the other special tendencies mentioned above in modifying the tendency to contract under disagreeable and expand under agreeable stimuli. While, therefore, this latter is clearly shown by this research as a real and strong tendency, it is at the same time shown to be only one tendency acting among many.

CHRISTISON.

THERAPY.

243. PARTIAL THYROIDECTOMY IN GRAVES' DISEASE. J. Arthur Booth, M.D. (Medical Record, 54, 1898, p. 217).

The author reports eight cases of Graves' disease operated upon with five cures—one died, in one no change occurred, one has been improved, and in this case the operation was performed only six months ago, so that the author expects further improvement and perhaps a cure, for the longer the period of observation after operation the better appear the results. The order of improvement was as follows: First the goitre diminishes; next the nervous symptoms disappear; then the pulse-rate and vasomotor phenomena improve, and the exophthalmos last of all. In fatal cases the deaths occur suddenly either at the time of operation or soon afterwards, and the rapid onset of acute symptoms, with death following in a few hours, has caused much speculation as to their cause. The author believes that cases of Graves' disease may be entirely cured by operative measures, and states that pathological and clinical evidence is in support of the view that the symptom complex is the expression of a primary neurosis multiplied by a secondary glandular intoxication. While the ultimate cause of the disease of the gland is still a matter of speculation, and a mortality of seven per cent. after operation is reported, he admits we cannot justly recommend it as a routine plan of treatment.

FREEMAN.

Book Reviews.

BEITRAG ZUR KLINIK DER RÜCKENMARKS- UND WIRBELTUMOREN
(Contributions to the Clinic of Tumors of the Spinal Cord and
Vertebræ). By Hermann Schlesinger, M.D., Private Docent in
the University of Vienna. Gustav Fischer; June, 1898.

It was the intention of the author to write a monograph on spinal tumors, but the work of Bruns, which appeared before this plan could be carried into effect, rendered such an undertaking unnecessary. Schlesinger, however, has had at his command the records of necropsies performed in the great hospital in Vienna, and his conclusions are based on the examination of a material which in value and extent can hardly be excelled. Thirty-five thousand necropsies, of which 151 were in cases of tumor of the cord or its envelopes, reported during the last eighteen years, afford an immense field of research. The volume of 209 pages contains a brief introduction, which is followed by chapters devoted to the pathological anatomy, etiology and clinical signs of tumors of the vertebræ and spinal cord. Fifty-six new cases are reported and 589 references to the literature are given. The book is well illustrated. Those who are familiar with Schlesinger's monograph on syringomyelia will expect to find a volume on spinal tumors equally well written, and we believe they will not be disappointed.

A tumor may arise externally to the vertebræ and grow into the canal through the intervertebral foramina, or it may develop within the bodies of the vertebræ, or within the canal and external to the dura, or within the membranes, or within the cord itself. Schlesinger finds that the vertebral tumors with consecutive involvement of the spinal cord are by far the most numerous, whereas tumors within the cord substance occur about as frequently as those within the meninges. In 400 cases of "intervertebral" tumors taken from the literature (by which we suppose tumors within the vertebral canal are meant) surgical interference could have been of benefit only in about 150, and if the vertebral tumors are added the percentage of operable cases is much lessened. Unusual forms of tumor are most common in the *conus terminalis*.

Tuberculosis of the spinal cord is treated quite fully, and we are informed that primary tuberculosis of the spinal cord has never been observed. The tuberculous growths resemble the syphilitic very closely, and the absence of the bacillus is by no means proof of the syphilitic nature of the process. Schlesinger has collected the reports of nineteen cases—including two new ones of his own and four rather doubtful cases—of gumma of the cord. He calls attention to the fact that in a number of cases in which the gumma was large secondary degeneration was almost entirely absent.

Considerable space is devoted to glioma, but the author's opinions on this subject are already well known.

Diffuse sarcomatous infiltration of the meninges, and even of the spinal cord, extending a considerable distance, is a recognized and interesting form of new growth. Schlesinger has collected the reports of thirteen cases of primary sarcoma of the cord without involvement

of the membranes; it is therefore a rare condition, whereas the primary sarcoma of the meninges and nerve roots is apparently the most common of the tumors arising in the meninges. Attention is paid to a number of rarer forms of tumor.

Primary carcinoma of the cord or its membranes is unknown, and metastatic carcinoma within the vertebral canal, not growing from a vertebra, is exceedingly uncommon. Even when the vertebrae are the seat of carcinoma the growth very rarely goes beyond the dura, and in those rare instances in which the dura fails to offer a barrier the cauda equina is the portion more often attacked. In the 35,000 necropsies the dura of the cervical and thoracic regions in every case prevented the further extension of the carcinomatous process. Primary carcinoma of the vertebrae probably does not occur. Kolisko has repeatedly shown that the primary lesion is elsewhere, and may be so small as to be easily overlooked. Schlesinger believes that carcinoma of the vertebrae cannot be considered especially common, and he speaks of a fact not generally known, viz., that primary bronchial carcinoma, itself rare, relatively often gives metastasis to the vertebrae. The most common primary seat of vertebral carcinoma is the mammary gland.

Schlesinger emphasizes the fact that cavity formation is found not only in the gliomatous, but also in the sarcomatous and tuberculous tumors of the spinal cord.

The cord may suffer a slight change in its form, from the pressure of a tumor, and this alteration is not always persistent when the tumor is removed. The cord is, therefore, compressible to a certain degree without being permanently injured. Circumscribed, even large, extramedullary tumors involve comparatively slightly the spinal roots, and this Schlesinger considers a clinical fact of great importance; vertebral tumors, on the other hand, do much damage to the nerve roots.

Schlesinger has not been able to confirm the statement of Charcot that arterial thrombosis is common when vertebral carcinoma exists, but he has found that thrombosis of the large veins of the lower extremities is not rare in cases of vertebral neoplasm. This explains why sudden death occurs frequently in vertebral carcinoma with embolus of the pulmonary artery as the direct cause.

The 35,000 necropsies show that the brain and its membranes are more than six times more liable to tumor formation than the cord and its membranes. Tuberculosis and glioma are relatively frequent in the different parts of the central nervous system, but gumma of the cord is much more uncommon than gumma of the brain. Secondary sarcoma is not uncommon in the brain, but is rare in the cord. A vertebral tumor involving the spinal canal and producing symptoms of nervous disease is thirty times more likely to be malignant than benign.

Spontaneous and intense pains are among the earliest, most persistent and distressing of the symptoms of vertebral carcinoma. Sciatica may be the only sign for a long time, as in one of Schlesinger's cases where it existed alone for two and a half years, or in another where it existed for two years.

The differential diagnosis of vertebral tumor may be most difficult, and caries of the vertebra may produce symptoms closely resembling those of carcinoma.

The indications and contraindications for surgical interference in neoplasms of the vertebrae and cord are given. We find that Schlesinger is not entirely pessimistic as regards the results of attempts at relief by surgical means. The mere opening of the vertebral canal, however, may be fatal.

SPILLER.

THE
Journal
OF
Nervous and Mental Disease

Original Articles.

ON THE ETIOLOGY AND PATHOGENESIS OF
THE POST-TRAUMATIC PSYCHOSES AND
NEUROSES.*

By JAMES J. PUTNAM, M.D.,

Professor of Diseases of the Nervous System in Harvard Medical School.

In spite of all that has been written about the post-traumatic psychoses and neuroses, the subject still presents to the searcher many fascinating problems for study and speculation, and others of highly practical significance, which need all the light that a careful scrutiny of our experience and a comparison of our observations can furnish.

What are the factors—for they are obviously multiple—that make these apparently trivial accidents the starting point of grave disease for some persons, while others pass through them unscathed; what part do the actual injuries of the brain and spinal cord play—the minute hemorrhages and necroses of which we so often hear—in shaping the future of the case; to what extent does the law create or increase the troubles for which it was meant to compensate, and what can the doctors do in this direc-

*Read in part before the Philadelphia Neurological Society, March, 1898. For discussion on this paper, see page 485.

tion, general practitioners or experts, to smooth the path of true justice and promote the real interests of the community and the dignity of their profession; what is the nature of these affections and what their proper treatment, and how far does ignorance, as regards these points, on the part of the profession and the community, tend to make a needless supply of nervous invalids, and what shall we do to improve the existing conditions?

These are a few of the practical problems that confront us, while problems of more purely scientific nature throng to the mind of every close inquirer.

A friend to whom I casually mentioned that I was writing on the traumatic psychoses said, "They don't exist. The cases are all manufactured by the doctors, who persuade their patients that they are ill, and thus make them so." Without fully sharing this drastic opinion, I do believe that in many of their features these cases belong largely to the preventable class, preventable by sufficiently judicious treatment on the part of physicians, and likely to grow less numerous as the community becomes better educated, and, as lawyers learn more and more to see and seek the best interests of their clients.

Let me say, however, at once, that while these diseases are obviously prolonged and made worse by the various baneful influences that cluster round the effort to obtain redress by suits at law, yet that would be a narrow judgment which should refer to this cause alone the fact that so many more bad cases of traumatic hysteria are seen in the law courts than on the football grounds.

That this fact is true I believe, but the "legal" causes, important as they may be, are by no means the only ones that can be adduced in explanation.

My personal experience is based on the examination of one hundred and eighty-two cases of patients who have consulted me for illnesses which, it was claimed, had resulted wholly or in part from accidents or injuries. Besides the record of these examinations, I have used the

notes of twenty-four additional cases of hospital patients, choosing the more striking and best reported cases of recent years. Many of these reports leave much to be desired as regards fullness, but they can be safely utilized for certain purposes. Of the private patients, one hundred and fifty-three consulted me with reference to claims for damages, while only twenty-nine came for advice as to treatment. All of the twenty-four hospital patients came for treatment alone, but five were prosecuting claims, or had prosecuted them in the past, and about the same number had, perhaps, made some sort of settlement, generally with an employer. Thirteen of the hospital patients had no legal claims to make. Of the two hundred and six cases, one hundred and thirteen were males; ninety-three females. The preponderance of males is, of course, due to their greater exposure, and no one can read over the histories without becoming convinced that the proportion of females would probably not have reached 45 per cent. but for a greater predisposition on their part of one or another sort.

In the interests of an analysis of predisposing causes, which will be given later, I have tried to divide the patients into four classes as regards their "social status." Had I been sufficiently intimate with them, I should have made the degree of *cultivation* and *mental balance* the basis for this division, but where, as was often the case, I could not form a satisfactory opinion on these points, I have divided the patients according as they earned their support by wages (Class I.); by small salaries (Class II.); by professional income, higher salaries, or established business (Class III.), or as they belonged distinctly to the leisure class (Class IV.). In Class I. there were sixty-two patients; in Class II., one hundred and seventeen patients; in Class III., twenty-five patients, and in Class IV., two patients. It would, perhaps, be better to group together Classes I. and II., as representing the patients who, in my opinion, based on such information as was at my dis-

posals, had had a relatively small amount of "social training," in the widest sense, and Classes III. and IV. as representing those who had had better opportunities in this respect. We should then have a Class A (I. and II.), comprising one hundred and seventy-nine patients, and Class B (III. and IV.), comprising twenty-seven patients.

For reasons which are partly obvious, but which will be discussed later at some length, the "medico-legal" cases fall mainly under Class A, the non-legal mainly under Class B. On the other hand, a division of the cases as regards indications of typical neuropathic predisposition, in the usual clinical sense of the term, shows that Class A contains, of neuropathic cases, 28 (18 per cent.), of non-neuropathic, 122 (80 per cent.); Class B contains, of neuropathic cases, 19 (73 per cent.), of non-neuropathic, 7 (36 per cent.). Here, again, the data are lacking in accuracy, but the general conclusion is probably fairly correct. Thirty cases were excluded from this latter estimation, leaving one hundred and seventy-six as a basis for the statement above given.

To classify as simply as possible the causes of the post-traumatic psychoses and neuroses, it may be said that the various agencies unchained by a sudden accident—whether they be nerve storms that are set up by a great fright, and go, as it were, crashing through the brain, or the emotions associated with memories and reflections after the event, or the impressions made without the intervention of consciousness, or, on the other hand, actual injuries, small or great—all resemble each other in that they all disturb, more or less profoundly, the normal hierarchy of the central nervous system.

This disruption of the old order may be followed either by a satisfactory establishment of the mental and nervous balance on the former lines, or by the formation of a new and necessarily unstable and unsatisfactory equilibrium on some other basis. The post-traumatic diseases may be defined from this point of view as expressions of the

various modes by which such a new equilibrium is reached. It is the expert's task to discover the laws that control this process of readjustment, and the recent studies into the psychology of the subconscious life furnish a valuable torch to light us in the search.

A long step was already taken when Dr. Hughlings Jackson, for a long time the foremost of medical psychologists, with keen instinct recognized, and with skill expressed the principle to which I have just referred—that in proportion as the nervous system, under the influence of disease, loses its power of working on its former basis, as indicated by symptoms of defect (or “negative symptoms”), it inevitably seeks to readjust itself to the changed conditions, and it is only the fact that the readjustment is often defective in practical efficiency which leads us to define its manifestations as disease (“positive symptoms”). What Dr. Jackson could not then realize for lack of sufficient data, was that, in this readjustment, subconscious processes of a high order and susceptible of study and classification greatly complicate the situation, making our analysis at once more difficult and more satisfactory.

In searching for the causes of post-traumatic psychoses we ought to include, on the one hand, all the influences that tend toward a disarrangement of the normal coördination of functions of the nervous system, and, on the other, all those that determine the form of the reorganization.

From this point of view I propose the following arrangement of causes in the order of their importance:

(A). Predisposing causes:—

1. Of social character.
2. Of neuropathic character.
3. Of toxic character (alcohol, syphilis, etc.).

(B). Causes operative from the time of the accident:—

1. Emotional shock.

2. The mental strain—not of emotional character—attendant on intense voluntary effort.

3. Painful or disabling injuries, such as sprains of the back or blows upon the head, not sufficient to cause deep loss of consciousness, yet sufficient to inhibit the voluntary control in some measure.

4. Injuries sufficient to cause deep loss of consciousness, such as powerful electric shocks or severe blows on the head, or severe concussions, such as presumably interfere with the intimate nutrition of the nervous system.

5. Slighter bodily injuries, even down to physical contact, if of a kind to excite or increase apprehension of danger.

6. Injuries of a kind to cause actual lesions within the central nervous system.

(C). The principal disorganizing causes operative after an accident are:—

1. Excitements and anxieties of diverse sorts.

2. The emotional excitements due to reproduction in memory of a past danger.

3. The continuance of pain, and the depressing effect of internal disorders, such as sprains, uterine displacements, etc. It is to be noted that the fact that such affections as these arise as the result of an accident often clothes them with a power to cause and perpetuate nervous symptoms infinitely greater than they would ordinarily possess. They become centres of widely reaching “association neuroses.”

(D). The influences that seem to me to take the principal part in re-establishing, on new lines, some substitute for the normal equilibrium disturbed by the accident are:—

1. Influences made possible by the impairment of innervation and vasomotor action due to the accident. Such are the causes of the various skin, vascular and organ affections and those of bacterial origin, and also of the

typical psychoses and neuroses. It is only the latter diseases that I shall discuss here.

2. Influences analogous to hypnotic "suggestion."

3. Influences equivalent to the formation of *habit*, "*irradiation*" symptoms, "*association*" symptoms, etc.

4. The emotional tendencies referable to the direct and indirect influence of lawsuits.

Some of these influences are important enough to justify further study of their mode of action. Those enumerated under A, B, C can be grouped primarily as (a) the influences associated with actual lesions and bodily injuries, and (b) those acting through the intermediary of the mind. I will begin with the first of these groups, but before going further with them I wish to point out that in the study of the traumatic psychoses hitherto the tendency has been to treat the strictly "traumatic" cases far too exclusively, though it is true that of late the reaction against this tendency has made itself felt, in some quarters, perhaps, too strongly. There is no radical difference between the hysteroid and neurasthenic conditions induced by a misfortune, or a surgical operation, and many of those induced by the mental and physical shock of an accident, and had this been borne in mind we should have heard less of the mistrust of the genuineness of all post-traumatic cases, and should have witnessed a more unbiased and systematic effort on the part of experts to sift the different etiological elements. Dr. S. A. Lord has reported, for example (The Boston Medical and Surgical Journal, June 23rd, 1898), two interesting cases from the records of our clinic at the Massachusetts General Hospital, as indicating how trifling operations may plant the seeds of very troublesome symptoms of just such a kind as follow accidents.

It often happens that exacerbations of neurasthenic conditions, or the outbreak of special symptoms of hysteroneurasthenic character follow special acute causes, the action of which is closely analogous to those of acci-

dent. This, indeed, every one knows; but it is not so well realized, I think, that slight degrees of hemianæsthesia and the like are fairly common in such cases, and that, altogether, their study may throw much light on the subject of the traumatic psychoses. For instance, a gentleman of my acquaintance had, after a severe attack of influenzal character, which eventually involved the frontal sinuses, an intense right-sided supraorbital neuralgia, for which he stayed a couple of days in bed. During this attack he observed that the sole of the right foot was moist, and tests showed a slight diminution of sensibility of the right foot and the right hand, while the surface temperature of the right foot was slightly greater than that of the left. This moisture lasted a few days, gradually fading away. There was no obvious difference of sensibility between the two sides of the face, though during the presence of the pain the right side sweated quite profusely, especially about the nose. No other signs of hysteria were present.

I have seen this same slight general hemianæsthesia in another case of facial neuralgia occurring in an apparently non-hysterical young woman, and in the cases of two men suffering from amputation neuralgia. Only by courtesy could these cases be called hysterical.

Since I began to look for them, I have seen several cases analogous to these which would ordinarily be called neurasthenic.

One case of this sort was that of a Jewish lady of middle life, seen in consultation a month ago. Although she looked in blooming condition and gave a history of past good health and gay spirits, yet, as a result of a series of slight domestic troubles, she complained of a variety of annoying symptoms, evidently based in part on hysteroid instability. Only by close questioning did she admit a slight motor difference between the arms, but on careful examination exactly the same conditions were found that characterize the light cases of traumatic

hysteria, a trifling but well marked difference in surface temperature and slight degrees of anæsthesia and analgesia, which I took great care not to produce by "suggestion," and which had obviously been unnoticed by the patient.

Sänger has shown, by his valuable study into the cutaneous sensibility of German workmen of the class that apply with especial frequency for pensions after injury, that such sensory disorders are not uncommon, due usually to alcohol, syphilis, previous injuries and similar causes.

Whatever opinion we may hold as to the relation between actual lesions of the central nervous system and the post-traumatic neuroses and psychoses, there can be no question but that accidents, even of moderate severity, such, for example, as often usher in the psychoses of hysterical type, are capable also of causing these *actual lesions*, presumably, as a rule, minute hemorrhages, but also vascular disorders and subtle nutritive changes. A good many such cases are on record, and I have myself seen a number, of which the following may serve as examples:

A lady, 38 years old, was riding in a buggy which was run into by a grocery wagon. The wheels of the two teams locked and the jar threw her out, although the buggy was not tipped over. She struck the ground with the left side of her face and head, but was not stunned, or only for a moment at most. On trying to rise, her arms and legs felt "numb" and powerless, and she could not stand alone or raise her hand to her head. The next morning the feet and legs seemed to be much better, but the hands were very painful, numb and prickly, and continued in this way for two weeks. She was not able to walk well for the first month after the accident, but the numb, prickling sensation left the feet in the course of the night following the accident. The micturition was all right from the beginning. The only indication of a girdle sensation was that on taking a long breath there was a sort of catch in the left side. She was unable to feed

herself for some weeks, and during the period that the hands were painful the flexor muscles of the fingers were the seat of cramps. The neck muscles were also slightly stiff. She was then able to walk pretty well and to use her hands, although slowly and awkwardly. All the tendon reflexes were exaggerated, and there was some incoördination of all four extremities, both static and motive, and a high degree of impairment of sensibility in the fingers.

No "hysterical" nervous symptoms were present at any time, and I take the case to be undoubtedly one of hemorrhage into the cervical enlargement of the spinal cord, induced without fracture of the vertebral column. I could cite three or four other cases of a closely similar sort, but pass to two where acute myelitis came on after injury.

One of these was that of a stalwart young man, who dove from a moderate height and became completely paralyzed, from the arms down, immediately after striking the water, though he did not strike his head upon the bottom so far as could be ascertained.

Neither of these cases was complicated by lawsuits.

The next case is that of a man of fifty-seven, who was thrown out of his buggy, in an electric car collision in a city street, so that he fell with some violence on to the sidewalk, and struck also upon the brick wall of an adjoining building. His knees were badly bruised, and for the first few days his legs felt numb and helpless. He then recovered his power of locomotion, but the "numbness" still remained, being especially severe along the under side of the thighs of both legs, the right being somewhat more affected than the left.

For two months after this he went about, but had considerable difficulty in getting up the stairs, and suffered continually from a soreness along the under surface of the thighs, so that the pressure of an edge of a chair caused him great discomfort. His feet felt as if resting

against a hot steam pipe. A week before I saw him, which was about three months after the accident, he had been attacked with a severe pain in the back, and this was followed by rapidly progressing paralysis of the legs and of the bladder, the loss of power becoming complete at the end of three days.

There was absolute immobility of both legs, including the motions at the hip joints, except that the motions of the left foot and toes were almost perfectly free. The knee-jerks were present, but not exaggerated; but the attempt to provoke them excited general twitching of the whole body. The sense of touch was slightly diminished, more so on the right side than on the left. A slight motion of the finger on the skin was felt at once and well localized. In proceeding up the leg I found that as soon as the knee was passed both contact and pressure were painfully felt. This hyperæsthesia was much greater on the inner side of the thigh than elsewhere, but was provoked by deep pressure all the way down to the foot. There was diminished sensibility to pricking, except where hyperæsthesia was present. The nutrition of the muscles at my first examination was normal. From this time the patient continued to grow worse, and the case developed rapidly into one of complete dorsal myelitis. After an illness of one year without improvement, he died, and I made a post-mortem examination. This showed extensive disorganization of the spinal cord, which was most severe at the level of the ninth dorsal nerve root, where the whole section presented a brownish yellow tint. The membranes were somewhat adherent, and covered with exudation, even as high as the cervical enlargement. The posterior and median columns showed evidences of secondary degeneration as high as in the cervical region, and some degeneration of the lateral column was seen at the fourth dorsal root and from there downward.

My object in reporting these cases is not so much that of adding to the clinical literature, as to show that in of-

fering the opinion, which I hold to be correct, that such lesions play only a relatively small part in causing the typical symptoms of the post-traumatic psychoses, I am not actuated by any disbelief in the power of moderate jars and blows to produce actual lesions as one of their results. Both the American and German periodicals of the past two years contain the records of carefully studied cases, showing the variety of the lesions producible by injury to be greater than would have been supposed. The list embraces not only hemorrhages and necroses, but diffuse alteration of blood vessels with the symptom-complex of progressive paralytic dementia—perhaps of vasomotor origin—and also lesions analogous to those of poliomyelitis. Injury may also give a strong impulse to the development of syringomyelia, and, perhaps, to that of all the degenerative affections. Can such lesions, however, be legitimately considered as a true cause of the psychoses and neuroses?

In the light of our present knowledge I think we may say that the only way in which this could happen would be through an inhibiting and deranging influence which the lesions might exert as centres of irritation. If it is true that profound derangement of nerve function may be excited in this way, then, in the course of the re-establishment of equilibrium, hysteroid psychoses and neuroses might readily emerge, just as in the case of the post-traumatic affections of emotional origin. It is indeed probable that irritations which cause pain, as, for example, strains of the back, which make every motion, even of the arms, an event to be dreaded and avoided, do act in this way, and that, as a result of the pain, the nerve functions of sensitive patients may suffer a widespread disturbance and inhibition, both locally and in a general sense. It is also theoretically possible that the same sort of inhibition should occur without the intervention of conscious pain or of any consciousness at all, and this view is strengthened by what we know of the relation of the subconscious to

the conscious processes, as underlying, for example, the outbreaks of hysterical convulsion. It is also true that hysteroid or neurasthenic groups of symptoms may be practically the sole manifestation of serious gross lesions, such as tumors of the brain, or may accompany such diseases as syphilis or the spinal sclerosis.

Again, it is reasonable to suppose that the action of disorders of general nutrition, such as those which attend and follow infectious diseases, and which are well known to cause or accentuate the psychoses and neuroses, find their analogue, in some measure, in the disorders of nutrition, due in part to actual lesions of the nervous system. This must at least be true in so far as such lesions interfere with circulation and digestion, and that which is true of these functions must be true of others which stand on the same plane.

These considerations make it appear probable that, to a high degree in exceptional cases and to a slight degree in many cases, actual lesions, even if minute and diffuse, do contribute to the occurrence of the post-traumatic neuro-psychoses. But, after all is said, it must, I think, be admitted that for the majority of accident cases this action makes itself but little felt, and is wholly subordinate to other etiological factors. Gross lesions, small or great, and whatever be their nature, usually fail to cause these hysteroid symptoms, and, in the great mass of cases—traumatic and non-traumatic—where such symptoms do occur, the main causes are psychical influences of one or another sort, acting on a nervous system which is, if the term be correctly understood, *predisposed* to such affections.

To be sure, cases are sometimes met with where the differential diagnosis as to the presence or absence of actual lesions is difficult or impossible, but with an opportunity to watch the patient we can at least soon tell whether, lesions or no lesions, the case is to have the relatively favorable issue that characterizes the traumatic hys-

terias, or the relatively unfavorable issue that characterizes the progressive degenerative processes. Even when the latter result occurs, however, we cannot be sure that actual lesions were the cause. A case with regard to which this doubt as to the existence of actual lesions arose is the following, which was, fortunately, not of medico-legal interest: A roofer, of good previous health, fell from the top of a building to the ground, 40 feet below. He was dazed, but not wholly unconscious. For three weeks he stayed in bed, suffering mainly from pain and soreness in the back and head. He then went back to work, but felt poorly, and had to give it up for a time every week or two. A few months later he had to give up entirely, the sense of soreness having spread all over the body.

I saw him first, at my office, two years after the accident, and found him weak, emotional and demoralized.

What especially attracted my attention, as possibly of spinal origin and due to actual lesions, were a prickling of the hands, which came on whenever they were placed in a constrained position, and at times spontaneously, and a gait slow and stiff, with scuffling of the feet at times on the ground.

Five years after the accident I received the following note:

"Dear Sir: In answer to your inquiry as to the state of my health, I will say that I feel very well, thank you.

"While I experience some of the symptoms of which I complained to you, especially at this time of the year, I am able to work at the roofing business. When springtime comes I feel like going to some place in the country and taking a rest.

"I attribute my improvement to regular living chiefly. It was since your treatment, and particularly your advice, namely, not to scrutinize myself so closely, and to make a firm endeavor to do a little work every day, that my greatest improvement is noticeable."

It should not be forgotten that the capacity of hysteria to produce symptom groups which would have been thought to occur only on the basis of gross organic change is being continually rated as higher and higher.

We have the "hysterical intention tremor" and the "hysterical paralysis agitans," and, according to Dr.

Prince's opinion, with which I fully sympathize, there is a hysterical form of neurasthenia.

Nonne has described a "pseudo-spastic paraplegia," with exaggerated tendon reflexes and ankle clonus, and Schuster has recently given his opinion that the kindred group of symptoms, associated with extreme rigidity of the back muscles, referred by Kümmel to disease of the vertebræ due to injury (a condition which certainly may occur) may be a form of hysteria.

I have recently seen a case of this sort, where the trotting of one or the other of the knees was so severe as to positively shake the floor of the room. The stiffness of the erector spinæ was so great in this case that the patient rose from his chair with great difficulty. Nevertheless, although this rigidity of the back muscles was so great, and the pain on forced movements so severe, leading one to believe that a serious strain must have been received, the patient asserted that his back had not troubled him much until after ten or twelve hours from the time of the injury, and the consideration of the case as a whole made the diagnosis of hysteria, or one of the many and varying affections which we cover by that name, highly probable, as covering the major part of the symptoms, at least.

But if it can be shown, even as a matter of presumption, that actual nerve lesions are rarely to be counted as direct causes of traumatic hysteria, let me not be understood as denying the indirect influence of physical violence, even in those apparently insignificant forms to which only in the technical dictionary of the law the name of violence could be accorded.

This question has become one of especial practical significance in Massachusetts since the recent decision of the Supreme Court, reaffirming a principle, which, though at first sight unjust and out of keeping with scientific doctrine, yet is doubtless based on practical wisdom; namely,

that one cannot recover damages for illness due to pure fright, unattended by personal violence.

If, however, a personal violence, no matter how slight, can be proved, even one which does not go beyond the degree of personal contact, then it is possible for the claimant to recover, not only for the effects of the violence or contact, but also for those of the concomitant emotion or other factors, provided, of course, it can be shown that the contact was a real cause of at least a part of the subsequent symptoms. In this state of affairs it is obviously important for experts to form their opinion as to how far trifling injuries, received under such circumstances as those assumed, may be real causes of subsequent symptoms.

The bodily injuries which tend to cause or increase emotion, in case of accident, are rather those of moderate severity than those which are surgically very serious. It sometimes happens, no doubt, that patients who have met with serious injuries are subsequently overcome by strong emotion, on looking back at the circumstances under which they were received, but serious surgical injuries in general, such, for example, as cause acute surgical shock, are not likely to increase the emotional tendencies of an accident. The case is different for moderate injuries, such as sudden jars or blows, and obviously for the reason that they disconcert the patient's will, impair his sense of confidence, diminish his power of self-control, and increase the apprehension of more harm to come, while they are not severe enough to induce the anæsthesia and indifference which attend prostrating wounds, fractures and dislocations.

These, then, are the conditions which make slight bodily injuries productive of harm, that they are received under such circumstances as render them capable of disconcerting the reason and the will, and creating an apprehension of greater harm to come.

The decision above alluded to was given in connection

with a case where a woman had claimed damages for nervous shock which was received while a drunken passenger was being put off an electric car and lurched slightly upon her during the process. The case was retried after this decision, and damages were awarded and allowed.

There is a subtle influence in a physical contact under certain circumstances that raises it to a high rank among the causes of nervous shock. It is easy enough to tell a rattlesnake from a carrot if it's by daylight and you're not in a hurry, but one would like to recover damages from the man who for joke's sake obliged one to make the diagnosis all of a sudden, in the darkness of the night, when he had just waked up from sleep, in a pitched tent on the banks of a Southern river. Of course, a trifling jar or a personal contact, even if experienced in connection with a railway accident, may remain a trifling matter; but this is not always the case.

In the instances just referred to the physical violence in itself was merely nominal. There are, however, other cases where, although slight, it has a definite effect of its own, though one which it is difficult to classify in terms of actual lesions of the nervous system. Instances of this sort are the jars and concussions received during railway accidents, where something occurs which is more or less analogous to what we class as concussion of the brain. Even here it is probable that in fact we have to do with a mainly psychical injury. To take a homely example, let one imagine himself given a violent shake by the collar while crossing a crowded street, where it was necessary to be on the lookout for rapidly moving teams. Such a shake, even if administered on the sidewalk, would have been disconcerting, but under the actual circumstances the effect might have been actually paralyzing.

Mental influences attending accidents, which tend to derange the normal equilibrium of the nervous system. These may be divided into (1) the *predisposing*, and (2) the *exciting* influences.

1. What is it that constitutes predisposition to the traumatic psychoses, and why is it that of two people sitting on the same car seat one is severely affected and the other not at all?

In some degree it is doubtless a constitutional neuropathic tendency, of hereditary origin, that causes this susceptibility, and many of the patients who become chronic hysterics and neurasthenics are obviously only working out their "manifest destiny." It is, however, distinctly my experience, and the same observation was, I believe, made long ago by Oppenheim, and more recently by Sanger, that in the great majority of the cases, including those where apparently trivial accidents have been followed by very serious results, no neuropathic tendency, in the usual sense of the term, can be detected. This is indicated by the analysis of my cases given at the beginning of the paper. In this estimation the diagnosis of "neuropathic" was admitted quite liberally, although the data were incomplete.

The best way to approach this problem of predisposition may, perhaps, be to consider what sorts of persons are relatively exempt from the severer forms of the post-traumatic hysteria and typical neuroses.

I choose out hysteria and acute forms of the typical neuroses because the arguments which I shall adduce do not apply equally well to some of the other post-traumatic affections. There are, for example, many persons of the relatively exempted classes who suffer from neurasthenic troubles, or from ill health in one or another form, which is often attributable to a combination of causes, of which an accident is one. Sometimes, indeed, the accident may have been the essential cause of the subsequent symptoms, but the fact that the patient struggled against the gradually rising illness with resolution and temporary success postponed the result, and made it impossible to assert positively that it arose from this cause alone. In most of these cases, however, occurring among the usually ex-

empted classes, the resulting symptom-complex is not that of typical acute hysteria or of mental affections bearing the hysteric stamp, but rather of neurasthenia or "association neuroses," or one of the types described so well by Prince in the Boston Medical and Surgical Journal, June, 1898.

Foremost among the exempted classes, in the sense thus defined, are those who meet with accidents in the way of sport, or of business of which accidents of a certain sort form a legitimate outcome, or of war. There are various good reasons why this should be the case. One is that the emotions which accompany such accidents are usually not of the depressive sort. These mischances are not associated, as a rule, with any sense of personal grievance, and do not fall with the heavy weight or startling terror of a misfortune wholly unexpected or unprepared for. The football player, or the artisan, discounts his injuries in advance. He knows that by watchful care he may prevent them, and that if he receives them he does but pay the price for his pleasure or profit; that he has no grudge to bear, *no lawsuit to bring*. Such injuries are, moreover, not associated in his mind with any exaggerated feeling of terror, of "social" origin. He has not grown up in an atmosphere of sentiment, shadowy and unnamed indeed, but intensified by hundredfold reflection, that such mischances are events to be deeply dreaded. He looks forward to recovery and more play or more work. Finally, the members of some of these exempted classes are young and spirited men. Yet it must be admitted, in view of the severity of some of the injuries which they receive, that if nerve lesions of small amount led often, of themselves, to hysteria, we should hear more of such results than we do hear.

Another relatively exempted class is certainly that of the men and women with highly trained self-control and cultivated intelligences. Whether from familiarity with the risks of travel or from the possession of a character

trained to accept philosophically the mischances of the world, or from habits of self-discipline inculcated by social training, they are little likely to let their reason and their will remain long dethroned.

Persons of this stamp are more likely to be found among the professional, the leisured and the higher business classes than among wage-earners and people of confined lives and small incomes. I do not, of course, maintain that this rule is an absolute one, or that fine and strong character is not as common among the poor as among the rich. Neither can I undertake to bring any great amount of statistical evidence in support of the opinion which I advance, since cases vary so widely that the numbers of any one sort, the severe hysterias for example, available for comparison as regards the social status or temperamental and intellectual training of the patients, are too small to be convincing.

I believe, however, that every physician of large experience, not to say every observant layman, must have gained the strong impression that, on the one hand, the ability to withstand the demoralizing effects of accidents and injuries is an indication of a good nervous system, and, on the other hand, that the sort of vigor thus implied is not incompatible with an excitable, even highly neuro-pathic, temperament, provided the social training has been of the appropriate sort.

The man who lives by his wits, the philosopher, the adventurer, the person who can look on an accident as a joke or a new excitement, or as a means of extending his experience; even the correct society man, whose rules of caste do not sanction a confession of weakness, and whom poverty is not staring in the face, are likely to pass unscathed through a railroad disaster which might seem an irreparable disaster to many a robust, hard working man of narrow experience and slender reading, spending to the limit of his earnings, and figuring, to be sure, on a life

of labor, but one free from cataclasms of such sorts as these.

If the people of the wider training do fall victims, I think, as I have said, that it is toward neurasthenic states rather than toward hysteria, that their symptoms tend.

I cannot trace out in proper order all the mental characteristics that impart such powers of resistance as I have in mind, or, on the other hand, all those which tend in the direction of diminished resistance. But I feel convinced that one good criterion is the degree of liability to be carried away by the contagion of "mob-madness," which has been studied so much of late. And here I feel sure that "*Cultur-Menschen*," be they never so neuropathic, have a distinct advantage. Of some of them it may be said that society has trained them into an army, giving them the power to resist panic that belongs to the disciplined soldier; of others that, in full knowledge of social laws and traditions they have chosen to disregard them, and so have gained in personal independence. Obvious illustrations of mob-contagion are sometimes seen in connection with accidents, and, as a matter of fact, a phenomenon of similar sort is very common. For every member of society is to a greater or less degree under the influence of the "social" opinion that an "accident", and perhaps especially a "railroad accident" or an "electrical accident" is an event to be greatly feared, and the ready yielding to this opinion is equivalent to a ready yielding to mob-influence.

It may appear incorrect to say that the poorer members of society are relatively unfamiliar with accidents, and, therefore, suffer from them unduly, since in many cases the very nature of their occupation exposes them to injury. But it is my belief that the chains of mental association are very closely drawn, and distinctions are felt at once by the instincts which the reason is often slow to define. The accidents which come to a man in the way of his business affect him as the injuries received in war affect the

soldier. In both cases a certain nervousness and timidity are apt to be induced for a time, but, as a rule, the power of resistance as regards that special form of danger improves as time goes on, while it takes a training of a more real character to make a person indifferent to dangers which have hitherto been unknown quantities.

I have tried, in these remarks on character and training as related to predisposition, to keep in mind the influences which could be exerted in cases not complicated by lawsuits, but one object was, of course, to point out that because we find, as we do, a larger number of severe hysterias among law-court cases than elsewhere, we should not jump at once to the conclusion that desire for gain is the main cause of this difference. It is a partial cause, no doubt, and it is possible to conceive of circumstances under which "football hysteria" should become more common than it now is, but it is also certain that the majority of the claimants for personal damages on account of personal injuries belong to a class of persons who are relatively predisposed to hysteria from injury, in the sense that I have mentioned, while, at the same time, their lack of fixed income, intensified by the "social" traditions which their dread of poverty has helped to engender, constitutes ample reason why they should feel obliged to go to law. It is easy, in view of these facts, to see why we find our traumatic hysterias in court, and why we do not find them in greater numbers in private practice, or even in hospital practice. I say "in greater numbers," because, of course, highly interesting cases, free from legal complications, are to be seen occasionally both in private and in hospital practice. The hospital list is the longer of the two, and there can be no question but that it would be longer still were it not that a stern fate supplies the vigorous tonic of forced work to the few of the hospital out-patient class who can find no one to go to law against, and that with this impulse they get relatively well, within reasonable periods of time.

It is a matter of practical importance, and at the same time a fact in confirmation of the importance of "social" causes, that not only does severe hysteria occur oftener among the classes of persons to whom I have alluded, but it is correspondingly slow to pass away, lasting often many years after a verdict has been rendered, in the "legal" cases.

In this connection the extreme hopelessness which characterizes one race of people, which have lately become very numerous among us, namely, the Russian Jews, is worthy of notice. Many of these patients cannot be reached by ordinary therapeutic means because they are impervious to encouragement. Their minds seem closed to appeals to hopefulness, and a preliminary training has to be used before much advance can be made.

I have expressed my belief that the legal complications, in these cases, intensify the illness and retard the recovery, but the question arises, Can we estimate the amount of this influence, and how soon does it become operative? When a person begins instantly, or within a few moments of an accident, to show the symptoms, not of simple nervousness and demoralization alone, but of typical hysteria, is it probable, or is it impossible, that the ferment of a desire for legal damages has begun to work?

I have seen a number of cases where the patients began to feel the familiar one-sided paræsthesia or tremor, or had nausea or vomiting, or hysterical rigidity, or other typical hysterical symptoms, from the moment of the shock.

Our own experience and the literature of genuine, non-legal cases of the fright neuroses show that this is just what we might expect to occur, and there seems at first sight to be no reason why we should adduce the new motive of a desire for gain unless from a gratuitous determination to see fraud everywhere where it might by any possibility exist. I think, however, it must be admitted that there is possibly a scientific justification for assuming subconscious ideas of certain sorts, of which the thought of

legal complications may form one, even from the first moment of the accident. We all carry with us, packed away in the depths of our minds, great numbers of prejudices and emotions of more or less fixed form, ready to spring out, as tigers spring on their prey when the keeper leaves the cage door loose. Many of these emotions are of "social" origin, and, indeed, in the course of our "social" training we all come, but some individuals and classes of individuals much more than others, to have a host of strong though vague feelings, all the stronger indeed for being vague, that arise in obedience to the word "accident," which we whisper to ourselves at the moment of a railroad collision.

It is not even necessary that we should whisper the word "accident" or "lawsuit," not even that we should definitely frame it in our thoughts. The word is indeed the focal point at which the ideas which give it its richness of meaning converge, but just as a blurred, and yet recognizable image is formed before the rays of light reach the focal point, so a vague conception may be formed before the word which symbolizes the complete conception has defined itself even in thought. There are few persons who would not find the emotions connected with a disaster strengthened if to the word or conception "accident" were added the word or conception "lawsuit," and I can readily believe that among those to whom I have alluded as being forced to go to law, if they can, whenever they get injured, the idea of a "lawsuit" and the vague sense of the need of "making out a good case" should exist subconsciously as a "social" conception of great power.

I cannot understand how in any more definite sense than this the intention of bringing a lawsuit can frame itself in the mind at so early a period or within the first few moments after an accident as to materially intensify or color the symptoms.

For completeness' sake I should next consider the toxic predisposing causes, such as alcohol and syphilis,

which Snger has studied so fruitfully. I must, however, omit this branch of the subject for the present, only pausing to express my impression that neither alcohol nor syphilis played a large role as a cause of illness in the cases of the vast majority of the patients whom I have seen privately.

The next group of influences to be considered—bearing in mind that we are still dealing only with the agencies which tend to break down the established equilibrium of the nervous functions, and to leave the patient a prey to the forces of disease—are those which *attend and follow the accident*. Of the first division of these influences I only care to remark that they cannot all be profitably grouped together under the head of “emotional excitement,” such as “fright,” although the tendency of late has been to do this, partly in the interests of a simplified legal presentation of the case. In many cases fright is obviously present; in others, it seems as obviously to be absent. It might be permissible to assume, for some of the cases of the latter class, that the physiological element in fright was operative, while the conscious element was absent, but this explanation is not of universal application. Fright is certainly not the only mental condition which exerts an injurious effect at such times. Reasonable anxiety, the exhaustion from physical and mental effort, the prostrating effects of pain, grief for fellow sufferers, are but some of the mental influences at work during and immediately after the accident, while others become of importance a little later.

The consideration of the second division of the influences at stake brings up the question, To what extent does the case receive its stamp from the events that cluster round the accident itself, and to what extent, on the other hand, may the symptoms be due to causes acting subsequently?

Probably we should all admit that the later influences are sometimes of great importance, and it is certain that,

even where no distinctly new ones come in, a disastrous amount of fright may arise from the recurrence in thought of events and dangers of the accident, which is lived over in imagination, just as it is so often vaguely lived over in dreams.

Many instances are on record where a veritable panic has occurred in this way, after all danger has passed. I bring up the point now only for the sake of calling attention to the difficulty of differentiating between these cases and those where the symptoms were initiated at the time of the accident, but appeared later after a lapse of hours or days. This latent interval received—I believe from Charcot—the striking name of “interval of meditation,” the meditation being, of course, subconscious, and used as indicating that a struggle was in progress between the old order and the new. There is always the danger attending the use of a picturesque expression like this that it will chain the imagination too closely, and, in fact, I do not feel wholly satisfied with Charcot’s term, and should prefer an explanation which let it be seen that in the interval before the complete establishment of the new set of symptoms the old order of things held on by a sort of momentum, which is only gradually overcome. A certain time elapses before the forces which normally control the working of the nervous system find out, so to speak, that they have lost their sway, but finally they yield with a rush.

The next question to be considered is with regard to the agencies (class D) which determine what form will be taken by the “*readjustment*” which follows the derangement due to the accident.

A number of interesting principles here come into play, the majority of which may be classified as follows:

I. It might be said that all persons—but some far more than others—carry with them latent tendencies to one or another of the typical psychoses or neuroses, which represent, as it were, natural planes of cleavage, taking the

form of disease. These specific affections stand ready to assert themselves in times of impairment of the normal innervation, just as the specific bacterial invasions are made possible by similar causes.

Thus it is that Graves' disease, or chorea, or even epilepsy, springs suddenly into existence after the shock of accident or fright.

This explanation would apply not only to the typical affections bearing, in their symptomatology, no close relationship to the injury—Graves' disease, for example—but it applies more than one might think to the typical post-traumatic neurasthenias and hysterias. Very often, to be sure, we have to deal, in these cases, with groups of symptoms forming no consistent complex, but representing the heterogeneous effects of panic and "suggestion" and acute exhaustion, and similar agencies, which impose as typical hysterias or neurasthenias, but are not really such, and do not run the same course with them.

The hysterias and neurasthenias following accidents are, in reality, susceptible of classification from two points of view; firstly, as representing simple deviations from health, the impression made by the patient upon the observer being distinctly that of an essentially unaltered individual, with his functions temporarily out of working order yet, in such a way that the character of the deviation from health exhibits specific features of one or another sort; secondly, as representing clusters of symptoms so compact in themselves and so different from the conditions that make up the stream of health, that they suggest separate organisms. This point is of importance, both as a help to classification and as an expression of belief as to the mode of origin of the so-called functional nervous disorders in general. We ought to accustom ourselves to speak and think of "hysteroid" affections and "hysteria" as related and yet distinct. The hysteroid affections may occur as aberrations of health, just as a healthy man may have a fright or a fit of passion; yet be-

tween hysteroid and hysteria there is only the difference that in the latter case the healthy influences have lost their pre-eminence so far that there is but little attempt at re-assertion. The impression made is no longer that of a diseased individual, but of an individual and his disease.

II. In the really typical cases it is probable that a certain degree of predisposition is always present, but it has frequently been suggested that some of the symptoms met with in these cases are due to an influence analogous to what is called "suggestion in the waking state."

In Dr. Sidis' recent book on the psychology of suggestion some interesting experiments and observations are recorded with regard to suggestion in the waking state, which are very apposite to this case in hand, although the distinction between the two sets of phenomena is probably less radical than his account suggests.

He shows that, in contradistinction to post-hypnotic suggestion, the waking suggestion succeeds best when, first, the patient is prepared by being plied with indirect influences, all pointing to the final end in view, and then at last the effective suggestion is given of a sudden, and in a way to be acted on at once. These conditions are in a measure paralleled in the conditions presented by the traumatic psychoses. The preparatory influences are represented by the disquieting dread of accident, acquired through social intercourse and the newspapers and constituting the "social predisposition" as indicated above; while the final suggestion is represented by the events of the accident itself, by which the patient is suddenly demoralized, and through which he receives, as it were, the command, "now go and be an invalid," or "be unable to use your arm," etc.

In accordance with this theory, many of the symptoms of the post-traumatic neuroses and psychoses, taking the form of pains, of disorders of sensibility or of motion, of mental depression with a tendency to recurrent dreams, of hypochondriasis against which the patient often struggles

in vain, are due to the working out of impulses resident in a mental life which is apart from the ordinary consciousness but plays the part of its "demon." In a similar way the nutritive processes may be affected, and that to a degree to which the patient's conscious volition is incapable of affecting them.

We are not justified in applying strictly to these subconscious mental processes the laws with regard to emotion that are derived from conscious introspection. It is well known from daily observation that a person outwardly calm may inwardly be deeply stirred. A depressing experience which one strives, and with apparent success, to thrust out of the mind, may really remain and prevent sleep at night or excite unpleasant dreams, and no satisfactory measure may be present to consciousness as to what the outcome of the half-felt or unfelt emotion will be. Just as an event, apparently forgotten, may flash into the mind without obvious cause, so these states of the subliminal or ultra marginal consciousness may come up when least expected.

It is probable that the half-dazed state into which a person is liable to be thrown by fear or by the complex influences attending an accident is peculiarly favorable to the lodgement of these subconscious fixed ideas.

Perhaps we are hardly justified in asserting that in those cases where consciousness is instantly lost, as from an electric shock, the capacity for the reception of even subconscious impressions is retained, but it is probable that even here the loss of consciousness is not necessarily to be taken as a warrant that all the mental powers have been abolished. Just as a person may wake up crying as from an unpleasant dream, and yet be wholly unconscious that he has dreamt, so he may suffer every degree of impairment of consciousness, in consequence of an accident, from simple confusion to entire unconsciousness, and yet retain the power of suffering all the results of an emotion, whether regarded as a physiological process or as a basis

for "suggested" ideas. Not only is the state of mind of many patients during the early moments of an accident equivalent to the half-hypnotized condition, but there are not a few persons who remain for days and weeks in an unnatural—usually excitable—condition of an analogous sort, in consequence of which they are not reliable judges of their own mental state and are the prey of unfavorable influences. Such persons often say that they feel calm; that they are not being influenced by the thought of an approaching trial, etc., while in fact the reverse is the case.

III. Several other principles which are important in this connection have recently been discussed by Dr. Morton Prince (Boston Medical and Surgical Journal, June 2d, 9th, 16th, 1898. "The Pathology, Genesis and Development of the More Important Symptoms in Traumatic Hysteria and Neurasthenia") with much clearness and method. Dr. Prince shows that *fatigue* may be a purely psychical phenomenon, a shadow of real, toxic exhaustion; and that, similarly, *pain* may arise in consciousness with extraordinary readiness with certain persons, or with many persons in abnormal mental conditions, just as other persons have a remarkable aptitude for the reproduction of visual or auditory images. Eventually, these and all such sensations may, by pure repetition, become *habitual* recurrences, like sensations of hunger. Again, many painful or distressing feelings, such as pain in the forehead on use of the eyes, and pain or paræsthesia in the head or back from exertions of any sort, may arise through an *irradiation* or *diffusion of energy*, which goes on with especial force because of the irritable condition of the nerve centres. Such pains might be called "intention" pains after analogy with "intention" tremor.

Morbid association, whereby cerebral events which have once occurred in juxtaposition tend forever after to recall each other, explains the persistent recurrence of vast numbers of special fears and complex mental states, conscious or subconscious. At first this chain of associated brain

processes is made up of a relatively small number of links, but there is a strong tendency toward a progressive widening of the vicious circle and a continual encroachment upon the healthy mental processes.

The first morbid event (as the circumstance of the accident) forms the centre of a sort of vortex, which gradually absorbs a larger and larger number of the cerebral reflexes within its influence, until the patient's whole mental life seems to centre on this single experience.

This tendency has been clearly pointed out by Dr. Mary Putnam Jacoby, in an instructive paper (New York Medical Journal, June, 1898. "A Suggestion as to Suggestive Therapeutics.).

IV. "*Litigation Symptoms.*" There are no special forms of symptoms, to my knowledge, which deserve this name, but it is true that the desire—half conscious, half recognized—to make out "a good case" tends strongly to intensify the hypochondriacal condition of the patients who seek relief at law and the special symptoms which characterize each case. It is doubtless true that the law, which was intended as a benefaction, often defeats its object and becomes a source of misfortune and continued illness. On the other hand, the overestimate of this influence, and the failure to bear in mind the considerations such as I pointed out in the early part of this paper, often leads experts for the defense to do gross injustice in special cases.

244. UEBER ACUTE PSYCHOSEN BEI KOPROSTASE (Acute Psychoses due to Constipation). F. von Sölder (Jahrbücher für Psychiatrie und Neurologie, 17, 1898, p. 174).

Careful and critical histories of six cases are here presented in which acute delirium developed following long attacks of complete constipation. The intestinal auto-intoxication produced the clinical picture of an acute maniacal excitement which persisted for from eight to fourteen days, and which with proper treatment in some cases resulted in recovery; in others weakness of the heart developed and the patients died in the acute delirious condition. Anatomically hyperæmia and œdema of the brain, congestion of the lungs, parenchymatous degeneration of the kidneys, heart and liver were found. The author presents the various views to account for the conditions and concludes that the auto-toxic theory seems to accord best with the facts.

JELLIFFE.

ON RESECTION OF THE GASSERIAN GANGLION,

By W. W. KEEN, M.D., LL.D.,

WITH A PATHOLOGICAL REPORT ON SEVEN GANGLIA REMOVED BY PROF. KEEN,¹

By WM. G. SPILLER, M.D.

ABSTRACT.

This paper (with nine colored plates) was written as a contribution to the three volumes published in 1898, in commemoration of the twenty-fifth year of Prof. Durante's teaching in Rome.

Dr. W. W. Keen said that he had done eleven operations for the removal of the Gasserian ganglion, and had reported six of these cases. (Transactions of the Philadelphia County Medical Society, 1894; The Medical and Surgical Reporter for March, 1894; and The American Journal of the Medical Sciences for January, 1896.) The results in these six cases are as follows:

Case I. The mental condition of the patient is not good, and he still has pain, but not the old tic.

Case II. The pain returned in six months, and still continues, although it is not so severe as it was before the operation.

Case III. The patient died in a week from avoidable septic infection.

Case IV. The patient was well at the end of three years.

Case V. The patient has been well for two years and a half.

Case VI. The patient has been entirely well for four years and a half.

Of the five other cases not heretofore reported, the following very brief résumé was given:

¹Read at the twenty-fourth annual meeting of the American Neurological Association, May, 1898.

Case VII. Removal of the Gasserian ganglion, after two prior operations, with portions of its roots; recovery; cure for two years and five months. No return of the severe pain, but paræsthesia.

Case VIII. Removal of the Gasserian ganglion as a primary operation; possible tear of the cavernous sinus, hemorrhage controlled by packing; recovery; cure for one year and eight months.

Case IX. Removal of the Gasserian ganglion after one prior operation; wound of the cerebral vessels while making the flap; rupture of the cavernous sinus, bleeding controlled by gauze packing; coma and hemorrhage, followed by death in three days; post-mortem examination was refused.

Case X. Death from shock in ten hours.

Case XI. Removal of the Gasserian ganglion after six prior operations; recovery; cure for sixteen months; eyesight lost from secondary corneal ulcer, caused by patient's neglect.

The ganglia removed from the last seven cases, V. to XI., inclusive, were handed to Dr. William G. Spiller for examination.

In Case VIII. no prior operation had been done. As all three branches were involved in the disease, the ganglion was removed as the primary operation. In six cases, VI. to XI., an attempt was made to remove the entire ganglion, and the illustrations showed perfect success in Cases VI. and XI., and reasonable success in the others.

Four points were then considered. First: Should the Gasserian ganglion be removed? This question, the speaker said, must be answered in accordance with (a) the mortality, (b) the result of the operation as to cure, (c) the possibility of avoiding the dangers of the operation. The mortality was shown to be about 22 per cent., much larger than should attend modern surgical operations. As to permanency of cure, there are only four cases in over a hundred operations in which pain has returned—one of

Rose, one of Dana, and two of Keen—but in Case I. of Keen the old tic did not return; no microscopical examination of the fragments removed was made. In Case II. the microscopical examination revealed no ganglionic cells or nerve fibres; both of these, therefore, were imperfect operations. Allusion was made to one case of Krause in which the sensory root was found diseased, and the pain returned on the other side of the face. The speaker said that the danger to the eye can now be eliminated by methods to be mentioned later. In view of the mortality, which is sure to be diminished when further experience has been obtained, and of the great probability of permanent cure, Dr. Keen believed the ganglion should be removed.

Secondly: To what extent should it be removed? He dissented from Tiffany's opinion that the motor root can be saved, and from Tiffany's proposal to remove the outer two-thirds of the ganglion, wrongly supposed to be in especial relation with the second and third roots, leaving the inner third.

Thirdly: Should the ganglion be removed as the primary operation, or should it be deferred as the final operation? He emphatically approved at the present time, of the removal of the ganglion as the final operation, partly in view of the danger, partly in view of the fact that there is a possibility of the return of the disease, even after excision of the ganglion. He urged, therefore, that if, after three or four months, drugs had not cured the affection, that the surgeon should do the operation before the ganglion was involved, without waiting longer in the hope of arresting the disease.

Fourthly: A few points in technique were considered, of which the only one necessary for mention here, perhaps, is the advice for the preservation of the eye. At the time of the operation the eye should be disinfected, and the lids sewed together at the middle by two or three stitches. Each day the eye should be carefully syringed

by a warm boric acid solution, and at the end of four or five days the stitches should be cut, and the eye protected by a Buller shield, i. e., a watch glass held in place by a rubber plaster.

Dr. Spiller reported the findings in the seven Gasserian ganglia examined by him. In six cases the lesions were very evident, but in the seventh, Case VIII in which no peripheral operation had been done, the portion of ganglion obtained for examination was nearly normal. The findings in the more advanced cases consisted of greatly swollen medullary sheaths and swollen axis cylinders, atrophied nerve fibres, empty nerve sheaths, atrophied ganglion cells, proliferated connective tissue, and sclerosed blood vessels. In one case the overgrowth of connective tissue within the ganglion and the destruction of nerve cells were very marked.

Dr. Spiller discussed the peripheral origin of trifacial neuralgia, and expressed the opinion that the relief of pain for one, two or more years, after peripheral resection of the nerve, was indicative of the peripheral origin of the disease. The Gasserian ganglion is not divided anatomically into thirds, and if it is true that the lesions originate within the ganglion, peripheral resection would not remove the irritation transmitted by the other branches of the fifth nerve, and we should not expect a diminution in the pain by removal of one of the branches of this nerve.

Dr. Spiller spoke of the normal condition of the sensory root of the ganglion removed intact by Dr. Keen. Krause had found the sensory root diseased in one case, and in this patient the pain had returned on the opposite side of the face after excision of the ganglion. The speaker referred to the contrast afforded by the normal sensory root in this case examined by him, and the abnormal condition of the fibres of the peripheral branches in this same case, within the ganglion. As peripheral and central nerve fibres of the ganglion arise from the same ganglion cells, Dr. Spiller thought it would be difficult to explain this

contrast afforded by normal and degenerated fibres, if the morbid process were primarily within the cells of the ganglion. As far as he was able to learn by an examination of the literature, the condition of the sensory root had only been reported in the two cases mentioned.

The abnormal condition of the sensory root observed by Krause warns us that in some instances the pain may return, even after removal of the ganglion, and there is abundant evidence now that pain may result from central lesions.

The speaker said that there could be no doubt that intense alteration of a chronic inflammatory character may be found within the Gasserian ganglion in certain cases of tic douloureux, and also that the sensory root may be intact in such cases. This, it seemed to him, was a satisfactory explanation for the relief of pain experienced by many patients in whom the ganglion had been removed.

Dr. Spiller called attention to the fact that if it could be shown that reunion of the sensory root does not occur after resection of this root, division of the sensory root might have the same remedial effect as excision of the ganglion, and be attended by a much lower mortality. He spoke of the desirability of making investigations on animals with this end in view.

DISCUSSION.

Dr. John Punton, of Kansas City, asked Dr. Keen whether he thought the operation of removal of the Gasserian ganglion was contraindicated in a woman of sixty-five with organic heart disease. In the case he had in mind, two minor operations for the relief of facial neuralgia had already been performed, with only temporary benefit.

Dr. Charles K. Mills considered the paper by Dr. Keen and Dr. Spiller the most valuable that had ever been made conjointly to the surgery and pathology of this important subject. Dr. Keen's surgical experience in connection with the subject, and his deductions therefrom, would certainly prove of great value to the neurologist and the general practitioner.

As regards the pathology of the disease, the speaker said

he felt inclined to differ somewhat from the views presented by Dr. Keen and Dr. Spiller, although he hesitated to do so, as those views were founded upon an actual examination of specimens. Personally, he was inclined to the opinion that *tic douloureux*, in the typical form in which we commonly see it, was a degenerative process of the peripheral sensory neurons. He did not regard it as a neuritis, in the ordinary sense of that word. In all the cases reported in the paper, with the exception of one, previous operations had been performed, and in every instance, except in this one, extensive disease of the peripheral processes near the ganglia was found. In six of them extensive disease of the Gasserian ganglion also was found. We may, in some of these cases, have to deal with a primary degeneration of the cell body; with an ascending neuritis of traumatic origin, from the previous operation; and with involution. A strong argument in favor of the theory that we have to deal with a true peripheral neuritis is that when a piece of the nerve is removed, the patient is free from pain for one or two years, or even for several years; but, opposed to this argument, we have the fact that in the vast majority of cases the pain returns.

Dr. C. L. Dana said that with regard to the pathology of this disease he was inclined to agree with Dr. Spiller. Dr. Mills, it appeared, was anxious that we should not look upon the condition as an inflammatory one. The term degenerative neuritis is often used loosely, and without any special reference to the inflammatory condition. Dr. Dana said he was inclined to believe that Dr. Spiller's specimens and investigations confirmed those of Dr. Putnam and himself, and that in most of these cases we have to deal with a degeneration of the nerve. Dr. Spiller's view, that its primary origin was in the periphery, was very well worked out. The speaker thought, however, that we should not look at the matter entirely from a microscopic standpoint. *Tic douloureux* is a disease not only of the nerve, but is an expression of general exhaustion, or the onset of degenerative changes. Some persons recover from it by proper rest and change and the use of certain restoratives. In a number of cases it is a self-limited disease.

The speaker said he felt under obligation to Dr. Keen for having presented the surgical and therapeutic sides of this subject so fully. His personal experience, however, in connection with surgical interference in these cases had been very unsatisfactory, and had strongly inclined him against such measures. He had the impression that surgeons now do not claim to be able to do anything more than to relieve the pain for a period varying from six to eighteen months. Dr. Dana said that, perhaps, his prejudice in this respect arose

from the fact that most of the patients he had seen were "bad cases," who had all been cut in one way or another, and generally were worse off for it in the end. A surgical operation done in the early stage of the disease may actually precipitate the malady, and aggravate it. The speaker said he could understand how a clean-cut operation in these cases might relieve the symptoms for a long time, but many operators did not do that kind of work; they left a lacerated nerve stump behind, and it was not to be wondered at that Dr. Spiller found degenerated axis cylinders.

As regards the removal of the Gasserian ganglion, Dr. Dana said that the value of the operation was established up to a certain point, but he had found patients very reluctant to submit to it.

In conclusion, the speaker said he had great faith in the value of toxic doses of strychnia in the treatment of tic douloureux.

Dr. Ira Van Giesen stated that he had made a number of examinations of resected nerves, including portions of the fifth, and had found an obliterating endarteritis of the vessels accompanying the nerves, similar to that reported by Dr. Dana. The speaker said he looked upon the disease of the vessels as the primary cause of the changes in the nerve fibres. After nerve section we may be sure that connective tissue will appear at the severed ends, with cicatrization and a gradual return of the symptoms.

Dr. Keen, in closing, said the question was purely a clinical one. Any operation which could give relief in tic douloureux, he thought, was justifiable, even in patients with an organic heart lesion; in such cases extra precautions were necessary in giving the anæsthetic. He did not agree with Dr. Dana regarding the unsatisfactory results of operative interference in these cases; his own experience had convinced him to the contrary. In the cases where he had operated the patients were free from pain for periods varying from one to three years, and he was inclined to believe that if an early peripheral operation had been done, the results would have been still better. He agreed with Dr. Dana that in some cases the use of massive doses of strychnia was very efficacious.

Dr. W. G. Spiller, in closing, said that on account of the reasons given he was much inclined to believe that this disease was usually originally in the peripheral fibres. He had examined the supraorbital and infraorbital nerves removed by Dr. Keen as a primary operation, in a case of trifacial neuralgia, and had found them much diseased. Pain had not returned when the patient was seen a number of months after the operation.

Clinical Cases.

A CASE OF CEREBRAL ATAXIA AFFECTING CHIEFLY THE RIGHT UPPER EXTREMITY WITH MARKED INVOLVEMENT OF THE STEREOGNOSTIC SENSE.

By INGERSOLL OLMSTED, M.B., TOR.,

Physician to the City Hospital, Hamilton, Ont.

The patient, H. P. D., act. 27 years, a clerk, was referred to me by my friend, Dr. A. B. Osborne, of this city, on the 20th of July, 1898. Dr. Osborne states that the patient first consulted him on the 11th of last May, and complained of double vision. When the patient was coming from the station he had difficulty in selecting the proper sidewalk, and avoiding people. I have taken the following extracts from Dr. Osborne's notes.

"May 11th. The pupils are equal and act consensually to light and with convergence. Right vision = $\frac{6}{6}$: L. V. = $\frac{6}{18}$ with correction = $\frac{6}{6}$. Under mydriasis; R. V. = $\frac{6}{36}$ with correction = $\frac{6}{6}$. L. V. = $\frac{6}{24}$ with correction = $\frac{6}{6}$. Fundi, normal. Outward movement of right eye restricted. Cover test shows outward movement of readjustment.

"May 30th. Cover test shows no movement of readjustment, with colored glasses can only induce diplopia by looking to extreme right and upward.

"June 3rd. No diplopia.

"July 14th. Patient thinks the vision of his right eye is failing. R. V. = $\frac{6}{15}$ with correction = $\frac{6}{12}$. L. V. = $\frac{6}{15}$ with correction = $\frac{6}{6}$. No change in the right fundus to account for the failure of vision.

"July 20th. Fundi, normal. Outward movement of right eye limited. Homonymous diplopia when looking to the right. Vision R. = $\frac{6}{12}$ with correction = $\frac{6}{6}$. Vision L. = $\frac{6}{12}$ with correction = $\frac{6}{6}$.

"No history of lues could be elicited, yet he was given specific treatment. By the 3rd of June the diplopia had entirely disappeared, but returned on the 14th of July. The following day the patient felt a numbness in the thumb, index and middle fingers of the right hand, also a prickling sensation, intermittent in character, beginning in the right lip and extending from there to the eye and ear of the same side. Owing

apparently to the numbness of his fingers he found difficulty in writing."

His history, taken July 20th, is as follows:

Father died at 65 years of Bright's disease. Mother died when 66, cause unknown. Both parents had been troubled somewhat during life with rheumatism. One ancestor two or three generations back, he states, had been insane. One sister aet. 33, is very nervous; four others are living and healthy; three are dead, two died in infancy and one at 31 years of some intestinal disease, the nature of which the patient does not know. Two brothers are living and healthy; two are dead, one died in infancy and one at 13 years of paralysis following measles. No history of cancer or of tumor of any kind in family.

Previous history good. Does not remember having had any disease. He denies lues, but admits having had intercourse on three or four occasions. Three years ago when riding a bicycle, he had a collision with another rider going in the opposite direction, was thrown from his wheel, partially stunned, and received a contusion of one eye, but does not remember which one. He was sore for a few days, but felt no other ill effects from his accident. He says he has always lived carefully, uses alcohol very moderately, but tobacco in excess at times. Since July 15th he has felt the numbness in the right thumb and fingers previously described. The sensation in the right side of the face, he speaks of as a flush, and says it is a prickling warmish feeling beginning at the upper lip and extending to eye and ear. It passes off in a few seconds to return in four or five minutes. When his attention is drawn to it, he thinks, this sensation comes more frequently. He has never felt anything similar in his right leg. He is a well developed man of medium size, intelligent, answers questions promptly in a clear distinct voice, but appears slightly nervous when being examined. The skin is normal in appearance, and free from scars; no cicatrix on prepuce or corona glandis, no enlargement of the inguinal, epitrochlear or cervical lymphatic glands, no roughness on clavicles (except, perhaps, a little on left), ribs or tibiae. The auscultation of the cranium on percussion yields negative results. No tenderness over the nerves of the face or of the extremities; no affection of hearing. The tongue is protruded in the median line and its muscles are properly innervated. No affection of taste. The examination of the heart, lungs, and abdominal organs, is negative. The urine contains neither albumin nor sugar.

The eye movements in all directions are good, except slight lateral nystagmus on either side. The pupils are equal and respond normally to light and on accommodation.

The facial and masticatory muscles act normally; jaw-jerk present. Touch, pain, heat and cold sensation are normal on both sides of the face.

The Upper Extremities: The arms are muscular, movements free in all directions; no ataxia on this examination, or tremor. The power is good, and equal on both sides; the elbow-jerk and biceps-jerk are present. The sensations of touch, pain, heat and cold are unimpaired, except, perhaps, to a very slight degree in the thumb, index and middle fingers of the right hand. (The stereognostic sense was not tested at this time.)

The scapular, epigastric, abdominal and cremasteric reflexes are markedly exaggerated on the right side. There also appears to be some hyperæsthesia of the right face, arm, and trunk.

Lower Extremities: The legs are muscular and of equal power. The knee-jerk on the left side is present, but on the right side is very much diminished and can only be produced very slightly with motor reinforcement. No Romberg symptom, gait normal.

Sensation of both legs is normal as regards touch, pain, heat and cold.

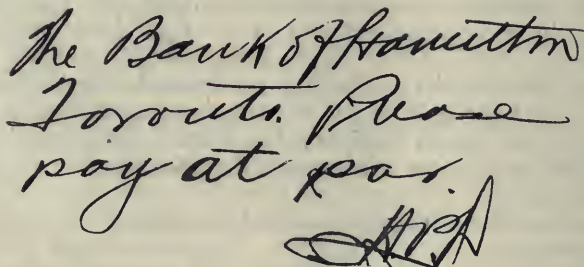
July 25th. The patient is in much the same condition, but complains that his hand and arm feel much stiffer, and the numbness affects the whole hand; when he buttons and unbuttons his clothes, the buttons feel as though they had been broken in two and only one-half remained. The right side of his face also seems sore and shaving has been painful. He says that the stiffness of the hand makes it very difficult for him to write, and when in my office he frequently opens and closes his hand, apparently attempting to remove the stiffness. He says that his right leg tires much more quickly than the left.

The electrical examination shows ready response of all the muscles to the faradic current.

Aug. 10th. During the past two weeks the patient has been getting worse. The condition of his eyes varies; at times there is diplopia, but at others none. The flush or paræsthesia of the right side of the face, is more marked, and extends, he states, down the side of his neck to the right shoulder and arm. There is well marked ataxia of the right arm and hand. When he brings his index fingers together there is difficulty in getting them to touch. When asked to touch his nose with his right forefinger, he makes the attempt with a certain stiffness and uncertainty, and usually touches the face at a point some distance to the right of the nose; with the fingers of his left hand, however, he executes this movement with precision.

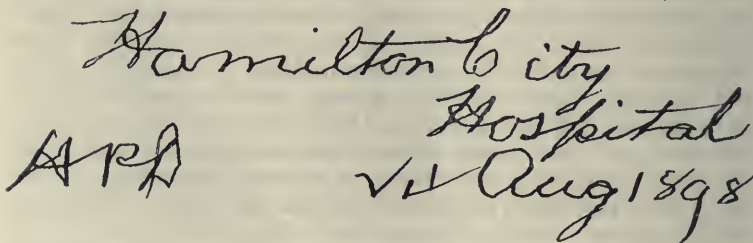
When asked to write, he takes up the pen very awkwardly, and has to use the left hand to fix it properly between the fingers of his right hand so as to get the point of the pen to come in contact with the paper correctly. The writing is done with slow, irregular, uncertain movements, and appears when finished quite unlike his former hand. The letters are tremulously and irregularly formed, have not the same slant, and the curves are made with difficulty. The more he writes the worse the writing becomes.

In Fig. 1 the handwriting of the patient before the onset of this affection is illustrated. In Fig. 2 is reproduced a sample of his writing at the date of this note.



The Bank of Hamilton
Toronto. Please
pay at par.
ARD

FIG. I.



Hamilton City
Hospital
ARD
✓ Aug 1898

FIG. II.

The movements of the right shoulder, arm and hand are all carried out with much less dexterity than those of the left. The muscular power of both sides, as measured with the dynamometer, is about equal. The patient says that not only the hand, but the whole arm and shoulder, has the stiff feeling.

Sensation to touch, heat and cold is perfect on both sides of the body, while that of pain is dulled in the right arm and on the right side of the trunk. The face, neck and leg on the right side are as sensitive to pain as on the opposite side. The most interesting feature of the case is the fact that the stereognostic sense is very much disturbed in the right hand. He cannot recognize a key, piece of money, or pen when placed in

his right hand, although they were recognized at once when placed in the left. He cannot button or unbutton his clothes with the right hand. He is able to tell a knife when placed in his affected hand. The farado-cutaneous sensibility is clearly diminished in the right arm, hand and on the right side of the trunk.

Aug. 17th. The patient remains in much the same condition except that the stereognostic sense is even more involved than on the previous examination. He does not know a coin, brush, scissors, pipe, ball or cube when placed in the right hand, but names them correctly when they are put in the left. He can give no information regarding the shape of the articles, but can, to a certain extent, tell their consistence when they are placed in the right hand. On testing the other parts of his body, as well as possible, using a book, a brush, rough and smooth surfaced articles, it is found that on his right arm and over the right half of the trunk, he cannot recognize a brush or an oblong block 2x3 inches, but does so on the corresponding parts of the left side. He recognizes a book the measurements of which are 3x6 inches, when it is placed on either side of the body. He can also tell a rough surface when it is placed on the right half of the trunk.

It appears that the stereognostic sense is thus almost completely lost in the right hand, and that it is to a certain extent impaired in the right forearm, right arm and right half of the trunk. This statement is also true for the muscular sense which was tested by means of different quantities of mercury in bottles. A bottle weighing three ounces feels to him as heavy as one weighing nine ounces. The position of the right arm, hand and fingers, when changed by the examiner, is, however, immediately recognized by the patient. All finer movements of the right hand and forearm are most clumsily performed, and the patient tends to drop articles placed in his hand.

It is also noticed that when he rises suddenly from his chair to a standing posture he has a tendency to go forward and a little to the right (propulsion). He states that he has noticed this tendency to go forward and to the right on two or three occasions during the last few days.

Dr. Osborne's note of August 17th says:—"Fundi, normal, no diplopia with colored glasses. R. V. = $\frac{6}{12}$ not improved by glasses. L. V. = $\frac{6}{12}$ with glasses = $\frac{6}{6}$. Cover test shows outward movement of readjustment. Pupils act normally.

"The fields of vision which are here given show marked contraction for white and red. Each temporal half is the one most involved."

We have before us, then, a well marked case of involvement of the stereognostic sense implicating principally the right hand and upper extremity. It is not associated with actual paralysis, but rather with a pronounced ataxia manifested by an inability to carry out accurately finer movements, such as those concerned in writing, buttoning, unbuttoning, and the like.

While disturbance of the stereognostic sense is by no means uncommon in cases of hemiplegia, the appearance of this symptom in the manner here reported, is, if the bibliography is to be relied upon, an extremely rare occurrence. As a matter of fact, there has been, thus far, no case to my knowledge reported in which a symptom-complex identical with that of the patient described above,

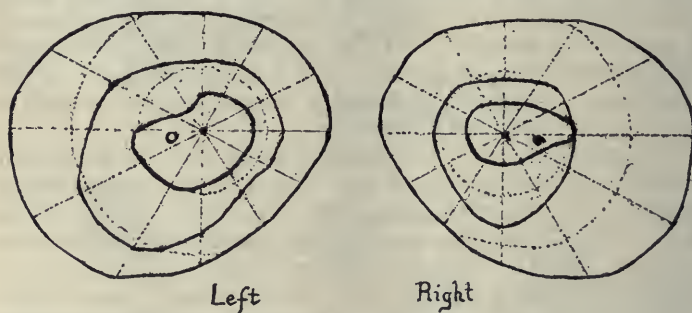


FIG. III. The innermost circles represent the boundaries of the red fields; the middle circles those of the white.

and with a similar mode of onset (absence of recent trauma) has been observed.

The cases approaching the one here described are (1) the classical case described by Wernicke¹, (2) the case recently described by von Monakow², and (3) the case reported by Burr,³ of Philadelphia.

In Wernicke's case there had been traumatic injury to the skull, with involvement of the region of the arm

¹ Wernicke, C. *Arch. a. d. psych. Klinik in Breslau* Leipzig, 1895, 2. p. 235.

² Von Monakow, C. *Gehirnpathologie*. Wien, 1897, p. 410 et seq.

³ Burr, C. W. *A Case of Psychic Anæsthesia*. *J. of Nerv. and Ment. Dis.*, xxv, p. 37.

area of the cortex on the left side. The case of von Monakow was also one of trauma followed by trepanation. In Burr's case there had been a blow upon the skull in the tenth year of life, followed by transient paralysis of motion and loss of sensation, and by permanent disturbance of the stereognostic sense. At the time of his report the patient was 24 years of age, and on grasping things in his pocket with the affected hand the objects could not be recognized. Burr is inclined, however, to regard his case as hysterical in nature. It is interesting to note that all the cases thus far reported, including the present one, have been individuals under 30 years of age.

One might give an opinion, and probably a tolerably accurate one, as to the exact localization of the lesion in the case here reported. The exact nature of the disease can at present be only a matter of speculation. The writer prefers, however, to reserve his judgment with regard to both points. The case will be carefully watched and its future progress and termination subsequently reported.

I take this opportunity of thanking my friend, Dr. Osborne, not only for having referred the patient to me, but also for his notes on the case.

NOTE.—There was absolutely no disturbance of "word-seeing" or "word-hearing."

Society Reports.

NEW YORK NEUROLOGICAL SOCIETY.

Stated Meeting, Oct. 4, 1898.

Frederick Peterson, M.D., President.

DOUBLE ATHETOSIS FROM INFANTILE CEREBRAL PALSY.

Dr. L. Stieglitz presented a boy, sixteen years of age, the youngest of several children. All the others had been well at birth, but he had been born asphyxiated, although the labor was normal. He did not walk until five years old. At the age of seven years he had scarlatina, and after that time the peculiar movements which he now exhibits became worse. Soon after birth it was noticed that he moved his arms, legs and head in a peculiar way. After a few years the movements remained at a standstill until the attack of scarlatina. On voluntary motion, the movements are aggravated. His face shows a constant succession of grimaces. The peculiar movements affect the trunk, as well as the upper and lower extremities. The tongue and the eyeballs are free from these movements. The deep reflexes of the arms are lively, as are also the knee-jerks, but not more than in many healthy children. His mental development is very good, but his physical development is rather deficient. Close study of the movements led Dr. Stieglitz to believe that the case was one of double athetosis, due to infantile cerebral palsy, the result of a lesion occurring at the time the child was born asphyxiated. These cases rarely show any defective mental development. In cases of athetosis the lesion is probably usually in the lenticular nucleus, or in the corpus

striatum, and it is not surprising that there is no epilepsy or disturbance of the mental development.

DISCUSSION.

Dr. B. Sachs said that as he recalled similar cases that he had seen, he was forced to the conclusion that the one just presented was a most excellent illustration of the combination of the choreiform and the athetoid movements. The probability of a non-cortical origin was, of course, very great. That there was a direct relation between the typical spastic cases of infantile cerebral palsy and the choreic and choreiform athetoid cases he verily believed. In a number of cases he had found that peripheral restraint on these unruly members, kept up for months, had had a remarkably good effect.

Dr. Edward D. Fisher said that he now had under observation a boy of the same age, who presented almost exactly the same symptoms, except that they were not quite so severe. It seemed to him that the lesion was very apt to be subcortical. The absence of epileptic conditions did not mean necessarily that the lesion was not a cortical one. The lesion was certainly not an extensive one in the motor tract.

Dr. Joseph Collins said that he agreed entirely in the diagnosis made by Dr. Stieglitz, and he was glad to see such a typical illustration of this group of cases. He did not favor the name "athetosis," nor would he apply it to this case. Taking the case in its entirety he would look upon it as an example of the choreic form of infantile cerebral palsy. He was inclined to think that the involvement was primarily of the corpus striatum and quite as much so of the optic thalamus, and he based this opinion on the study of the patient's motions, coupled with his spastic gait. The question of the existence of epilepsy in such a case did not seem to him of much importance, for the opinion was growing yearly that idiopathic epilepsy is a cortical disease. He had tried peripheral restraint for months at a time without observing the slightest benefit, even temporarily.

Dr. Peterson thought the case was undoubtedly one of post-paralytic morbid motion, and he agreed with Dr. Collins that athetosis was not a proper name. The position was athetoid, but the movement was rather a polymyoclonus. The term "athetosis" should be applied to a slow, vermicular movement.

Dr. C. A. Herter remarked that he thought the term was applied to much more rapid movements, and it did not seem to him that there was a proper pathological basis for such a distinction as that made by the last speaker.

Dr. Collins thought the term "athetosis" should be limit-

ed to rhythmical movements, and "choreic" to non-purposive, dancing movements.

Dr. Stieglitz, in closing the discussion, said that in cases of infantile cerebral palsy all the different varieties of movements mentioned were often combined, so that perhaps it would be better to select a name which would cover all of them. When the boy is less excited the movements are usually less rapid, and more vermicular in character. Under ordinary conditions, he does not present a spastic facies. The restraint treatment had been advocated by Dr. Hammond when he had first described these cases.

DISSEMINATED INSULAR SCLEROSIS.

Dr. Joseph Collins: The patient who is before you has the symptoms of two rather obscure diseases of the nervous system, and I bring him here that you may assist me in determining the one from which he suffers. He is 41 years old, married, and by occupation a silk weaver. His personal history is that he has been a user of stimulants and tobacco, the latter rather intemperately. Physically, he has led a very active life, and in many directions, particularly fencing and boxing. He has been an amateur athlete. The only point of interest in his family history is, according to his own statement, that a brother younger than him by two years, complains of the same trouble in walking that he does. When our patient was 33 years old, he had a sore on the penis for which he was treated by Dr. Geo. H. Fox. The latter assured him that this sore was of the nature of a chancroid, and gave him only local treatment. Aside from bubo, which complicated it, this terminated uneventfully in a few weeks, during which time he was treated in private practice by Dr. Fox. Aside from the attack of the grippe, which he had in 1892, he has been quite well. His present illness dates back nearly three years, the initial symptom having been, he thinks, dizziness, which was sufficiently great to interfere with his locomotion. Apparently this vertigo was not continuous, because he says that when it would come he would have to hold to something to prevent him from falling. About this time he must have had some trouble with

his sight, for it appears that he went to the New York Eye and Ear Infirmary, where he was told that he had locomotor ataxia. The symptoms that next appeared after the dizziness seem to have been what he describes as piercing pains in the calves and the sensation of heavy weights in the thighs. So far as I am able to infer, this constitutes the major part of the patient's complaint until the beginning of the present summer, although it should be said that he has been sexually impotent since two years. The fact that he has been unable to work has, according to his own statement, preyed very much upon his mind and made his symptoms worse. About six months ago his bowels and bladder became functionally derelict, and sometimes the one or the other would discharge their contents without his being aware of it. At this time, likewise his speech and handwriting became affected, both in very much the same way as they are now. That is, the enunciation is measured and scanning, the intonation rather high and uniform, while the handwriting is very irregular and ataxic. About the same time, the patient avers that he became uncommonly emotional. He would burst into tears at one time, and at another would grin or smile senselessly. Accompanying these physical symptoms, the patient underwent a psychical alteration. He says that he became very forgetful, that he was unable to read understandingly, even the newspapers. This state persists in a very much slighter degree to-day. For instance, it is quite impossible to elicit the symptoms of his disease except by asking him leading questions, and he forgets easily. At present, he is able to concentrate the attention sufficiently to read a newspaper or magazine understandingly.

His complaint at the present time is of difficulty in locomotion and impaired dexterity. The abnormalities of articulation and the slight psychical aberrations do not concern him sufficiently to complain of them. The functions of the bladder and bowels are now more nearly nor-

mal, although occasionally there is some incontinence, especially of urine. He does not complain of pain, nor of tight band sensation about the waist, and the feeling as if there were heavy weights in the thighs has disappeared. Examination shows a man of medium height and good muscular development, whose body is the seat of a marked deformity due to a cervico-dorsal scoliosis which he believes to have been caused by carrying heavy loads in the right hand or on the right shoulder during boyhood. As to the plausibility of such an explanation each one must determine individually. Both feet show the deformity which is known as pes cavus, pied bot of the French writers. The plantar surface of each foot is very much hollowed beneath the arch, while all the toes are forcibly extended save the first phalanx, which tends to claw the floor. When the patient stands, titubation of the whole body is the most striking phenomenon, and this persists, though to a lesser degree, when he sits. The gait is ataxic and reeling; unlike the ordinary taboid patient, he does not watch his feet while walking. He is rather more inclined to fix some point at or near his objective, and keep his vision on it until he reaches it. Station is extremely uncertain when the feet are approximated, and when he closes the eyes under such circumstances he is pretty sure to fall. The superficial, or cutaneous reflexes are present. The tendon jerks of the lower extremity are all absent, while of the upper extremity they are all present. The right palpebral fissure is somewhat larger than the left, and to explain this the patient says that it followed an attack of sore eyes. The pupils are of medium size, slightly asymmetrical, and do not respond to light. When the backgrounds of the eyes are viewed with the ophthalmoscope the left optic discs appear rather pale, a state of pink atrophy. The right optic nerve is normal. Tactile and thermal sensibilities seem to be normal. There is well marked analgesia over both legs. The patient has been examined four times only

with great care, but the analgesia seemed to be present on each occasion, although varying somewhat in intensity. There is no perversion of sensibility in any other part of the body. Color sense is normal, and the visual fields are unchanged. Senses of smell, taste and hearing are intact. The trouble with speech and writing have been already sufficiently referred to above.

Now, the question arises, has this patient Friedreich's disease, or has he multiple sclerosis? Perhaps, indeed this question is not sufficiently comprehensive. Some may be inclined to ask, and legitimately, I think, can diseases of the cerebellum be eliminated on the one hand, and genuine tabes on the other? Personally, I am inclined to the opinion that the case is one of disseminated insular sclerosis, although perhaps the evidence pointing to such a diagnosis is not so direct or convincing as some might wish. If a brief analysis of the symptoms be made, it will at once be seen that there exists a certain number of conditions which are usually considered inimical to the diagnosis of Friedreich's disease. These are, first, the patient's age; second, the sensory disturbances; third, the perversion of function of the uro-genital sphere, and fourth, loss of the pupillary light reflex and paleness of the temporal side of the optic nerves. On the other hand, all of these, and the other symptoms and physical accompaniments which the patient presents, including the deformities of the trunk and foot, can be explained by the existence of islets of sclerosis in the posterior columns, in the oblongata, and possibly in the cerebellum. If my diagnosis be the correct one you will agree with me, I think, that it is one of the most atypical thus far reported.

DISCUSSION.

Dr. Sachs said he was of the opinion that the case was probably a somewhat atypical example of multiple sclerosis. There was after all not so much difference between this case of multiple sclerosis and the cases of combined sclerosis that one sees, for he had observed cases of combined sclerosis

which at one stage had presented all the symptoms of a spastic paralysis, and subsequently the appearance of typical cases of tabes.

Dr. Fisher said he would agree with the diagnosis of disseminated sclerosis, basing it largely on the mental condition and the character of the speech, particularly the former.

REMARKS ON THE KIDNEYS IN CASES OF CEREBRAL HEMORRHAGE AND CEREBRAL SOFTENING.

Dr. Christian A. Herter read a paper with this title, in which he gave some of the results of his observations in 464 autopsies on adults at the Presbyterian Hospital. There were twenty-three cases of cerebral hemorrhage, eleven of cerebral softening and one of softening and hemorrhage. The three cases of hemorrhage in patients with large kidneys showed the lesions of chronic diffuse nephritis. Six of the patients with small kidneys were males, while all the patients with large kidneys were females. Of the patients with large kidneys, four had cerebral softening, two thrombosis and two embolism. The great majority of the medium kidneys were granular. In the cases of cerebral softening the kidneys presented a granular surface and freely stripping capsules. According to this series of observations, cerebral hemorrhage was most frequent among persons with small granular kidneys, and least frequent in those having large kidneys. None of the cases of hemorrhage had smooth kidneys, on the other hand, the majority of the cases of cerebral softening were associated with smooth kidneys and freely stripping capsules. Where there is a high arterial tension and the urine is of low specific gravity and contains very little albumin, and few, if any, casts, it may be inferred that the kidneys are small and granular, and this condition, in cerebral cases, points strongly to hemorrhage.

DISCUSSION.

Dr. Mary Putnam Jacobi said that she had recently seen a case which bore on the relation of uræmia and hemorrhage. The patient was a woman of about forty-five, who had been previously in excellent health. She awoke one morning to

find that her vision was extremely dim. The next day she complained of double vision, and a paresis of the abducens appeared and increased until the muscle became completely paralyzed. The urine on several examinations was found entirely free from albumin, and granular casts were found without the use of the centrifuge. The urine contained a large quantity of urea. The dimness of vision was probably uræmic and the paresis of the abducens due to a very minute hemorrhage in the sixth nerve.

Dr. Herter said that it seemed to him probable that in Dr. Jacobi's case the symptoms were due to hemorrhage rather than œdema, although the latter should always be borne in mind. It was a common thing to find considerable urea during the uræmic state. This was probably to be explained by a gradual accumulation of the urea in the blood, owing to the defective action of the kidneys, and this accounted for the fact that these patients often pass a normal quantity of urea in the urine at the time that the symptoms of acute uræmia are present.

TRAUMATIC HÆMATOMYELIA, WITH REPORTS OF CASES WITH AND WITHOUT AUTOPSY.

Drs. Pearce Bailey and P. R. Bolton presented a paper on this subject from which extracts were read by Dr. Bailey. Speaking of that class of cases in which the hæmatomyelia is consequent upon injury to the spinal column, the authors said that in a large proportion the general destruction of nerve tissue is so severe that hemorrhage can have had but little influence. The blood is poured out first and most profusely into the gray matter, and this central limitation is often very sharply defined. The columnar extension of the blood is usually in the gray matter of the anterior or posterior horns, and the column extends further upward than downward. Occasionally it may extend into the white matter just behind the middle commissure. These hemorrhages are most commonly found at autopsy between the fifth cervical and the third dorsal segments. The lower dorsal or lumbar cases either do not come to autopsy or else only after so long a time that it is difficult to draw definite conclusions regarding the pathological findings.

The evolution of the lesion in spinal hemorrhage is somewhat obscure. The natural tendency is to absorption of the blood so that cavity formation is an early consequence of hæmatomyelia. A symptom of some importance is a band of thermo-anæsthesia extending a little distance above the general anæsthesia. In primary hæmatomyelia bleeding into the substance of the cord constitutes the primary lesion. There are two forms, viz.: (1) the localized; and (2) the disseminated. A force sufficient to rupture the blood vessels of the spinal cord must affect the nerve elements, hence when bleeding is prominent it is reasonable to expect lesions of the nerve cells. In the majority of cases, however, the hemorrhage is the most important factor. The focal form of primary hæmatomyelia is always found in the lower cervical or upper dorsal region. Most of the cases result from falls. One form of injury which is especially fertile in producing this injury is a sudden bending of the neck, such as occurs in falling of heavy weights on the neck, diving in shallow water, etc. The symptoms differ from those of crushes of the cord, in which the white and gray matter are both implicated. The paralysis is at first flaccid everywhere, and the reflexes generally are either diminished or lost. An injury to any part of the spinal cord above the upper lumbar region, severe enough to cause paraplegia, will usually inhibit or abolish the knee-jerk for a time. This is eventually followed by an exaggeration. In the muscles of the lower extremities the paralysis usually changes within three weeks from the flaccid to the spastic type. Immediately after the accident the paralysis of motion is usually wide-spread and severe. Sensibility to touch is little, or not at all, modified in primary focal hæmatomyelia; there is, however, analgesia or thermo-anæsthesia, or both. In the cases reported the power to distinguish between heat and cold was lost while sensibility to pain was unaffected. Compared to the prognosis of secondary cases of injury, the outlook in

primary focal hæmatomyelia is favorable. Primary disseminated hæmatomyelia occurs after severe general traumatism. In these cases capillary hemorrhages were found throughout the cerebro-spinal axis. Microscopic examination shows small masses of red blood cells lying, for the most part, in the gray matter, but also in the white matter. They are not accompanied by other evidence of destruction, and are not artefacts.

DISCUSSION.

Dr. Sachs said that there seemed to be every reason to suppose that in many cases which were classified as of secondary origin there must have been effusion, not only into the cord, but around the cord. On this account, the question arose as to whether many of the symptoms might not be due to compression of the root fibres. He had seen dissociation of sensation as one of the earliest symptoms of Pott's disease and Pott's paralysis. It need not, therefore, be a symptom of destruction of the gray matter. He recalled a series of cases that were not traumatic in origin, were rather slow of onset, and occurred in adults, and yet presented all the typical symptoms described under the head of hæmatomyelia. The question arose as to whether in the non-traumatic cases the peculiar arrangement of the blood vessels in the spinal cord might not account for some of the cases in his series. A case now under observation was instructive for the reason that the lower extremities alone were affected, and there was an association of the spastic symptoms with a dissociation of sensation.

Dr. Onuff said that he had seen the case just referred to by the last speaker. The patient stated that soon after a tiresome bicycle ride he noticed that he could not urinate easily. The next morning there was a tingling sensation in the legs, but it was not for five days that a partial paraplegia appeared. At this time, incontinence of fæces was also noticed. There was atrophy of the leg affecting chiefly the adductors of the right thigh. The sensation of pain was exaggerated, so that there was a marked drawing up of the limb when it was pricked. The difficulty of urination was followed by incontinence. The atrophy in the thigh would seem to indicate a higher lesion, while, on the other hand, the initial bladder and rectal symptoms would point to a lower lesion.

Dr. Fraenkel said that he thought the abolition of the reflexes in such cases as those described in the paper was of much diagnostic importance. Enough cases had been re-

ported to show that even in the face of the absence of the reflexes it was possible for a communication to exist between the upper and lower portions of the cord. He regretted that the condition of the deep sensibility had not been recorded in the cases quoted.

Dr. Bailey remarked that in one case in which the reflexes were absent, the muscular sensation had returned.

145. UN CAS DE SURDITÉ VERBALE PURE TERMINÉE PAR APHASIE SENSORIELLE, SUIVI D'AUTOPSIE (Word-Deafness Terminating in Sensory Aphasia; Autopsy). Dejerine and Sérieux (Journ. de Méd., Feb. 6, 1898).

In reporting this case the authors say that only four cases have been recorded of what Lichtheim called sub-cortical word-deafness, and what Dejerine has proposed to call pure word-deafness. In this condition the patient has lost simply the comprehension of spoken language and the power of repeating and writing to dictation.

The present case is one of the four and was reported by Sérieux in 1893. After that date the patient gradually grew worse and the speech difficulty became extended into a general sensory aphasia. The autopsy, with careful microscopic examination, revealed no sign of a focal lesion, but a general atrophy of the temporal convolutions, bilateral, and diminishing in degree from above downward. This atrophy was caused by changes strictly cortical, and involving primarily the cells, the more superficial ones most. The authors sum up, briefly, as follows:

1. This autopsy definitely settles the question of the localization of pure word-deafness, showing it to be due to a lesion exclusively cortical, consisting in this case of a chronic poli-encephalitis, while in the case of Pick the lesion was both cortical and sub-cortical. This is the first case of the kind that has been shown to be due to a purely cellular lesion.

2. The case shows, as does that of Pick, that pure word-deafness is due to a bilateral, temporal lesion, located in the ordinary auditory centre.

3. Considering this localization, it appears probable that in pure word-deafness we have to do, not with a separation of the auditory centre from the centre of recognition of words, but with an impairment of the functions of the former. This opinion is corroborated by the fact that this patient, although she preserved the sense of hearing for a long time, gradually lost it.

4. The gradual and progressive transformation of pure word-deafness into a sensory aphasia is to be emphasized. For a considerable time the patient possessed intact the faculty of internal language, and it was only by degrees that the auditory centre for words became involved and that then appeared alexia, "jargona-phasia," and paraphasia.

PATRICK.

Periscope.

With the Assistance of the Following Collaborators:

CHAS. LEWIS ALLEN, M.D., Wash., D.C. R. K. MACALESTER, M.D., N.Y.
J. S. CHRISTISON, M.D., Chicago, Ill. J. K. MITCHELL, M.D., Phila., Pa.
A. FREEMAN, M.D., New York. H. PATRICK, M.D., Chicago, Ill.
S. E. JELLIFFE, M.D., New York. JOSEPH SAILER, M.D., Phila., Pa.
WM. C. KRAUSS, M.D., Buffalo, N.Y. HENRY L. SHIVELY, M.D., N. Y.
W. M. LESZYNSKY, M.D., New York. A. STERNE, M.D., Indianapolis.

CLINICAL NEUROLOGY.

146. DRITTE MITTHEILUNG UEBER DIE PAROXYSMALE, FAMILIAERE LAEHMUNG (A Third Communication Concerning the Paroxysmal, Family Paralysis). S. Goldflam (Deutsche Zeitschrift für Nervenheilkunde, xi, 1897, 3 and 4).

Goldflam has been able to observe this peculiar disease in another family, and in several additional members of the family he previously reported. One case may be given in detail. The attacks began in the eighth year of life, and occurred once or twice a year until the seventeenth year, but from this time until the present (twenty-second year) they have occurred monthly, or at shorter intervals. They usually begin in the evening with general weakness and drowsiness, and while they pass off during sleep, as a rule, they may result in complete paralysis, which lasts twenty-four to forty-eight hours, or longer. Improvement usually begins in the evening, and motion is restored within a few hours. Consciousness, speech, deglutition and the action of the sphincters, are not affected. The pain experienced during the attack is probably the result of the complete immobility. Occasionally the weakness begins acutely. When the weakness is felt in the morning hours the patient can sometimes ward off the paralysis by active movement and massage. Peculiar changes in the electrical reactions are noted. A copious meal seems to produce an attack. Goldflam observed complete and flaccid paralysis of the muscles of all the extremities and neck in this man, but the movements of the face, tongue, eyeballs and throat; the functions of the bladder and rectum; the general sensation, and the special senses, were not affected. Examination of the gastric contents revealed nothing abnormal. The most important etiological factor is the family predisposition. The changes in the electrical reactions of the nerves of the face, between the attacks, shows that the cranial nerves are not intact. There seems to be some relation between the paroxysmal family paralysis and dystrophia muscularis progressiva, for Bernhardt has observed these diseases in a father and son. Changes in the muscular fibres (rarefaction and vacuolization) were observed by Goldflam in a

number of cases, and were not limited to one family. They resemble those described by Dejerine and Sottas in myotonia congenita. In the second family, which Goldflam reports, the three eldest children were affected.

SPILLER.

147. ENTERALGIE (Enteralgia). Potain (La Clinique, Jan., 1897).

In a clinical lecture on the above-named affection, the author first remarks that abdominal pain is in no wise of diagnostic importance as it is common to a host of divers affections, and consequently the first step in the examination must be the exclusion of all organic disease, including tabes, the painful crises of which may closely simulate enteralgia.

The positive symptoms of the latter disease are recurrent attacks of severe, paroxysmal, cramp-like pain, generally beginning in the right hypochondrium, and following the course of the transverse and descending colon, coming on without apparent exciting cause, without reference to the ingestion of food and accompanied by characteristic and peculiar stools. For a day or two preceding the appearance of pain the stools become hard and their evacuation difficult; as the colic appears and continues the stools are progressively smaller and may become ribbon-like or about the size of a lead pencil. With the cessation of the paroxysm their calibre gradually increases, and a copious evacuation or even diarrhoea terminates the cycle. An attack lasts from a few hours to several days, and during its continuance the sigmoid flexure may be distinctly felt as a hard cord that is sore but not exquisitely tender to pressure. There is complete anorexia, emesis is frequent and may be biliary, moderate tympanites is generally present, as is also rectal tenesmus with frequent desire to defecate.

The two important causes of the affection, which is to be considered as a neurosis, are a neuropathic disposition and the uric acid diathesis—*l'arthritisme*—the gouty, rheumatic tendency. As the essential condition is intestinal spasm, in the treatment purgatives are to be carefully avoided, belladonna and opium being the proper remedies; but ether or valerianate of ammonium is also recommended, and hot baths may be tried. Between the attacks general measures alone are to be relied upon. Gastric purgatives, salines, aloes and senna are to be avoided. For constipation, castor oil, sulphur and rhubarb are preferred according to the author. Mild hydrotherapeutics constitute an admirable therapeutic measure, but bath or douche should never be employed when the patient is fatigued, for instance, after active exercise. If there be laxity of the abdominal parietes it is to be treated by vigorous and oft repeated faradism. The most important as well as the most difficult element of treatment, however, must be a perfect physical and mental hygiene.

PATRICK.

148. CASUISTISCHE MITTHEILUNGEN AUS DEM GEBIETE DER NEUROPATHOLOGIE (Clinical Communications in Neuropathology). M. Dinkler (Deutsche Zeitschrift für Nervenheilkunde, xi, 1897, 3 and 4).

1. Encephalitis acuta hæmorrhagica (?) recidiva.

A child of neuropathic ancestry, born in normal labor, had no signs of disease until he was two years of age. At this period he fell from a stool upon the occiput, vomited, had tonic and clonic convulsions in the right arm and leg which gradually ceased within two days, leaving a right-sided hemiparesis of a few days' duration. About a year later he had another slight fall, after which fever, un-

consciousness, vomiting, convulsions on the left side of the body appeared, and within a few days left-sided hemiplegia, lasting a week and a half was noticed. After the attack the child's mind was somewhat enfeebled and he had nocturnal enuresis. At the age of four years, after a fall from a step, he had a temporary renewal of his former symptoms. A few months later he had another attack with transitory hemiplegia and transitory amaurosis of both eyes without pupillary symptoms. The patient was received into the hospital during this last attack.

The diagnosis of tuberculous meningitis seemed improbable, on account of the transitory nature of the hemiplegia and the bilateral amaurosis, for the latter, Dinkler says, is not a sign of meningitis. The symptoms indicated at least two, and probably three, foci of disease. The motor tract could not have been destroyed. The lenticular nucleus is very vascular, and is a favorite location for encephalitis. It is probable that numerous small hemorrhages occurred in this body, or which is less likely on account of the speedy restitution, in the entire motor cortex of the right side. The amaurosis was believed to be due to lesions in the white matter of both occipital lobes, as its transitory character was not in favor of cortical destruction, or the absence of pupillary symptoms of thalamic lesions.

2. Syphilitic disease of the right frontal lobe with neuritis optica duplex præcipue dextra.

A man, thirty-one years old, complained of severe headache in the frontal and temporal regions, which was more severe on the right side at night. Typical mucous plaques, bilateral optic neuritis, involving especially the right side, right-sided mydriasis and disturbance of equilibrium with forced movements toward the left were noticed. The process was undoubtedly syphilitic. The lesion was believed to be a gumma, involving the optic nerves and the right frontal lobe.

3. Syphilitic vascular disease of the left Sylvian artery.

The symptoms in this case, transitory left-sided paralysis of the tongue and arm, fornication in arm and leg, etc., could be referred to lesions of the right parietal and temporal regions and basal ganglia. The most interesting features were found in the peculiar sequence of the symptoms, and in the right-sided homonymous hemianopsia, with right-sided optic atrophy and hemianoptic pupillary reaction.

SPILLER.

149. ARRET DE DÉVELOPPEMENT DU MEMBRE SUPERIEUR CONSÉCUTIF A LA TRAUMATISM DATANT DE L'ENFANCE, ATROPHIE MUSCULAIRE NUMÉRIQUE (Numerical Muscular Atrophy in a Case of Arrested Development of the Upper Extremity). Klippel (La Presse Médicale, 62, 1897, p. 49).

Klippel gives the clinical history, and the pathological report in the case of a man who in consequence of an injury to his left elbow at the age of three years, had an arrest of development of the left upper extremity, and who died at the age of 54 from pulmonary tuberculosis. A number of measurements taken during life showed a considerable difference in length and circumference between the right arm and the left. The left elbow joint was completely ankylosed. The very careful and thorough pathological examination showed that there was neither diminution in volume nor change in structure in the histological elements in the affected extremity, but that there was a distinct diminution in their number. The palmaris longus muscle, when measured at the same points on the two sides, gave right, circum-

ference $4\frac{1}{2}$ cm., left $3\frac{1}{2}$ cm. The difference in volume was also evident in other muscles of the forearm. The individual muscle fibres were of the same size on the two sides, but on the left they were fewer in number. The pronator teres and pronator quadratus had disappeared on the left side, their usual position being occupied by an aponeurotic layer. The right median nerve contained nineteen bundles of fibres, the left sixteen, but the individual nerve fibres showed no degeneration. The bones on the left side were altogether smaller than on the right, but their structure was the same. The anterior horn of the spinal cord in the cervical region was smaller on the left side than on the right, and in one section of actual count there were found thirty-five cells in the right, only eighteen in the left anterior horn. In the brain, the right ascending parietal convolution was slightly narrower than the left.

ALLEN.

150. ALIMENTAERE GLYCOSURIE BEI KRANKHEITEN DES CENTRALNERNEN-SYSTEMS (Alimentary Glycosuria in Diseases of the Central Nervous System). Von Oordt (Münchener Medicin.-Wochenschrift, 1, 1898, p. 2).

The author examined the urine of 178 patients suffering from different diseases of the nervous system, both functional and organic, after having caused to be ingested in each case grape sugar—100 grm. dextrose—under suitable precautions against error. The Trommer, Nylander, Phenylhydrazin and Tenreatich tests were used and the sugar, when present, was determined quantitatively by the polariscope. He draws the following conclusions. Alimentary glycosuria occurs in a certain percentage of cases.

a. In diseases of the structures within the cranial cavity, and is here caused, partly by encroachment upon the "diabetes centre," partly by central disturbances of nutrition resulting from encroachment upon the cranial space, pain, psychical disturbances and different reflex processes.

b. In a group of functional neuroses, neurasthenia, hysteria and traumatic neuroses.

It does not occur in a number of other neuroses, in true epilepsy; generally not, in diseases of the spinal cord, where there is no involvement of the medulla. Alimentary glycosuria can pass into spontaneous glycosuria.

ALLEN.

151. IST DIE PROGRESSIVE PARÄLYSE AUS DEN MICROSCOPISCHEN BEFUNDEN AN DER GROSSHIRNRINDE PATHOLOGISCH-ANATOMISCH DIAGNOSTICIRBAR (Can General Paresis be Diagnosed with the Microscope). O. Schmidt (Allgemeine Zeitschrift für Psychiatrie, 54, 1897-98, p. 178).

The author presents an excellent résumé of the various pathological findings which have been described by various authors and which have been regarded as characteristic of the disease. He shows that hardly any of the various lesions are constant. Thus though the vessels are usually affected, they are not always diseased, and moreover similar lesions have been described in other affections. The sclerotic areas are not constant and are subject to much variation. The newer researches on the changes in the ganglion cells would seem to offer the best opportunities for definite conclusions, yet these have not been always corroborated, nor are they universal. The changes in the nerve fibres are inconclusive and the general conclusion would seem to be that a need exists to differentiate anatomical types and correlate if possible the clinical phenomena.

JELLIFFE.

Book Reviews.

GEHIRNPATHOLOGIE. I. ALLGEMEINE EINLEITUNG. II. LOCALISATION. III. GEHIRNELUTUNGEN. IV. VERSTOPFUNG DER HIRNARTERIEN. Von Dr. C. v. Monakow, A. O. Professor der Neurologie an der Universität in Zürich. Mit 211 Abbildungen. Nothnagel's Specielle Pathologie und Therapie. Bd. IX. Theil I. Wien, 1897 (A. Hölder). Pp. I—IX and 1—924.

This volume, from the pen of a man already famous for his researches in human neurology and the experimental pathology of the nervous system, Prof. C. von Monakow, of Zürich, merits more than passing attention. No matter upon what topic this investigator and teacher might choose to dwell, his subscription of an article would attract to it the attention of most neurologists, but dealing; as the book before us does, in large part directly with the field in which its author's own investigations and experiments have been most fruitful, its advent will, we feel sure, be everywhere greeted with enthusiasm.

The volume forms a part of the elaborate system which has for some time been appearing under the editorial supervision of Prof. Nothnagel, of Vienna. The system as a whole is an exceedingly strong collection of monographs, and really does much, along with a few others which could be mentioned, to redeem this method of medical publication from the disrepute into which it was fast being driven; for the superficial compilations, hurriedly thrown together, which, under the names of systems and handbooks, have been disseminating loose and indifferent medicine and worse rhetoric in this and other countries for some time past, bade fair to condemn it utterly.

Von Monakow's book, as its title indicates, is divided into four principal parts. Under the first heading, the General Introduction to Cerebral Pathology, he considers: *A*, the Anatomy of the Brain; *B*, the Physiology of the Brain; *C*, the General Pathology of the Central Nervous System, and *D*, the Clinical Manifestation of Organic Diseases of the Brain.

The sub-section dealing with the anatomy of the brain makes interesting reading. Although, from the nature of the book, of necessity brief, reference will be found to the principal well-established facts. After a short introduction, in which the phylogeny and ontogeny of the nervous system are considered, the author immediately passes on to a description of the morphological constituents, the principal centres and important bundles of fibres of the adult brain. The sulci and gyri of the pallium, the basal ganglia, the projection-fibres, commissures and association bundles all receive due attention. The account given of the diencephalon, mesencephalon and rhombencephalon is brief, but being entirely free from padding will be found sufficiently full for most of the needs of the clinician. The text is accompanied by simple but helpful anatomical illustrations.

The neurological histologist will perhaps complain of the small amount of space allotted to the "elements of the nervous system," and it must be admitted that, even bearing in mind only the needs of the practical man, this section might have been lengthened with advantage. One gains the impression, which is probably true, that von

Monakow has been more interested in experimental physiology and pathology, and the study of cases of secondary degeneration, than in the application of modern cytological methods to the investigation of the structure of the nerve cells.

The general architectonics of the nervous system are, on the other hand, very satisfactorily examined, and the author is to be congratulated upon his concise and clear presentation of the conception of the neurone-complexes—the grouping together of neurones to form special functional elementary mechanisms. This is preceded by a somewhat detailed account of the principal methods employed in the investigation of the finer cerebral architecture. The methods of serial sectioning, of comparative anatomy, of ontogeny, including the following of myelinization in serial sequence, the method of Golgi and its modifications, are in turn referred to. As might be expected, much stress is, and with right, laid upon the importance of the study of secondary degenerations in the tissues derived from human beings, who, during life, have suffered from lesions of the nervous system, which have been carefully studied at the bedside. It is by means of such studies and of similar ones upon tissues derived from experimental animals that von Monakow himself, following upon von Gudden, has been able to contribute so liberally to the general stock of neurological knowledge. The method of investigation based upon an examination of suitable instances of congenital malformations also receives its fair share of praise. It will be recalled that it was by this method that von Leonowa, working with von Monakow, proved that the spinal ganglia, with their ganglion-cells, and peripheral and central nerve fibres along with a complete muscular system, can become developed in the entire absence of the spinal cord.

The classification given by von Monakow of the gray matter, and of the white matter is new; the novelty consists, not in the distinction of the nuclei of origin of the motor and sensory nerves, and in the recognition of the nuclei terminales of the central axones of the spinal and cerebral peripheral sensory neurones, for these, since the embryological studies of His, have met with general acquiescence, but rather in the grouping of certain gray masses in the diencephalon, mesencephalon and rhombencephalon as cerebral appanages or dependencies (*Grosshirnantheile*). Under the designation *Grosshirnantheile* von Monakow includes all those structures which, though anatomically not always consisting of homogeneous masses of gray substance, still in their functions as well as in their whole economy (nutrition) are wholly or partially dependent upon the cerebral cortex. These gray masses are evidently phylogenetically young, for they are absent in lower forms and increase progressively in size and number in direct proportion to the development of the cerebrum. Here, in the first place, are classed by von Monakow the majority of the nuclei of the thalamus, and especially the corpora geniculata (lateral and medial) of the two sides. The substantia nigra, certain elements in the superior colliculi, pons and medulla also come in this category. This view, which, it may be recollected, has been more than once advanced on previous occasions by the same author in special articles, also assumes that the majority of white fibres extending between the cerebral dependencies and the cerebral cortex are corticopetal, not corticofugal in direction. Other neurologists have held this view with regard to the corpora geniculata, but the theory is not in harmony with prevalent conceptions regarding many of the thalamic nuclei. Should it prove to be true, as von Monakow apparently believes, that all of the thalamo-cortical axones are corticopetal in

direction, and further, that the bundle of axones from each nucleus of the thalamus is distributed to a perfectly definite, circumscribed and ascertainable cortical area, then the hypotheses held by many neurologists with regard to the nature and functions of the thalamus and with regard in general to the relations existing between the diencephalon and telencephalon will have to be materially altered. At present, however, it seems scarcely safe to deny the existence of every and any corticofugal connection between the pallium and the thalamus.

While von Monakow's treatise is on the whole remarkable for its accuracy and carefulness of statement, still one or two points to which exception might be taken are observable. In the first place, the bundle of medullated axones formed by the descending limbs of bifurcation of the central axones of the peripheral sensory neurones which correspond to the sensory portion of the N. trigeminus is spoken of as a rule as the "aufsteigende Wurzel," although the author is careful to say that the fibres arise from the Gasserian ganglion and that they terminate in the medulla. Why the majority of German writers, and it must be confessed also a goodly number of English neurologists, persist in designating as *ascending root* a bundle which they certainly know consists of *descending* fibres is difficult to understand. Any one who has taught the anatomy of the nervous system to medical students knows that the difficulties of neurological instruction, especially with regard to the direction followed by the axones of the various fasciculi, are sufficiently great even when the nomenclature applied is entirely rational. It is but little wonder that students resent being told that the axones of an ascending root descend! Might it not be well to do away then with the false term, especially since it can be replaced by the entirely satisfactory "*tractus spinalis nervi trigemini*."?

The description given by von Monakow of the vestibular nuclei in the rhombencephalon is, in the opinion of the reviewer, open to serious criticism. The *nuclei terminales* seem to be confused in the text (p. 82) with the *nuclei originis* of the nerve, a matter of little importance perhaps to a reader of much experience (for is not similar confusion omnipresent in neurological bibliography?), but most unfortunate for the less advanced student. Part of the vestibular nerve *arises*, von Monakow states, in the medulla; the nerve, he emphasizes, has *nothing* to do with Deiters' nucleus, but *arises* in the main from Bechterew's nucleus! He admits that a *part* of the vestibular nerve has its origin in Scarpa's ganglion. The report in this connection will, we feel sure, be materially altered in a second edition.

Objection might with justice be made to the description on p. 114, in which the so-called N. opticus is assumed to be homologous with the peripheral sensory nerves; and to that on p. 128, in which the path underlying the impulses concerned in visual perceptions is designated as "*zweigliederig*," but these are minor criticisms and too much stress is not to be laid upon them.

In sub-section B, the physiology of the brain is dealt with. After emphasizing the extraordinary importance of animal experiment for the investigation of the cerebral functions, a portion of the work which, by the way, might be read with advantage by those who are still in doubt as to the utility and justifiableness of vivisection, the author treats of the differences of function in the various gross subdivisions of the brain in different groups of animals. For though telencephalon, diencephalon, mesencephalon, metencephalon and myelencephalon are in a large series of animals to be regarded as derivatives of similar portions of the primitive medullary tube, yet

they are by no means always exactly homologous. On the contrary they differ markedly in the individual representatives of the vertebrate series, not only quantitatively but also qualitatively in degree of development, and in the significance of each of the parts for the neural economy. In this connection von Monakow calls attention especially to the experimental results of Steiner and Schröder on lower forms, and of von Gudden, Goltz, Horsley and Schaefer, Munk and others on higher forms. He concludes that the difference in behavior of animals after cerebral defect corresponds completely to the morphological differences in the foundations and memberment of the individual cerebral centres. The lower an animal stands phylogenetically, the less does the intelligence, sensation and locomotion of the animal suffer from removal of the cerebrum. If cerebral removal does give rise to symptoms of this sort, they concern in the lower forms first the intelligence and the spontaneous taking of food, and only later in higher forms (with gradually increasing intensity) are the individual senses injured, especially that of sight; serious disturbance of gross locomotion does not result from cerebral defect until the highest phylogenetic stages have been reached. Von Monakow, therefore, views with favor the theory advanced by Steiner when he defines a brain to be a general movement centre, in connection with the activities of a nerve of special sense. Wherever these conditions meet, there a brain exists. The higher the animal, the farther forward the centres of control (*Wanderung der Function nach dem Vorderende*). Von Monakow extends the conception of Steiner by utilization of anatomical and physiological studies, classifying the centres as "phylogenetically old" and "phylogenetically young." By the former he means the fundamental neural structure, already indicated in Amoebæ, which contains all the mechanisms necessary for primitive or "mechanical" life; this structure is characterized by the tolerably great functional independence of its parts (mid-brain, hind-brain, after-brain). By the latter he means the "supplementary" structure, represented by the cerebral cortex and the regions of the cerebrum dependent upon this and developed in direct proportion to it, namely, his *Grosshirnantheile*. This supplementary structure, which attains to its highest development in man, is further characterized by the principle of the strict subordination of the so-called *Grosshirnantheile* to the cerebrum, and the principle of the co-operation of several portions of the brain anatomically widely separated from one another.

For each of the organs of special sense there would appear to be two anatomically separated nuclei of reception in the cerebrum, one phylogenetically old, the other phylogenetically young. It is especially through the latter that the connection for the cerebral cortex is made. In man, for the reception of sensory pictures and for the majority of complex reflex acts which are acquired through the intervention of consciousness, it is the phylogenetically young structure which is active. In lower forms the young and old centres are very incompletely differentiable anatomically, while in the lowest vertebrates the phylogenetically young centres appear to be entirely absent. Thus in connection with the optic neurones, the mesencephalic terminations of the N. opticus would correspond to the phylogenetically old centre, while the diencephalic terminations (in the corpus geniculatum laterale and the pulvinar of the thalamus) would correspond to the phylogenetically young centre. In fish the whole N. opticus terminates in the roof of the mesencephalon; the diencephalic termination is met with first in birds (Edinger); in man the mesencephalic termination by means of the brachium quadrigeminum superius is relatively insignificant, while the terminations in the corpus

geniculatum laterale and pulvinar and the connections by way of these through the radiatio occipitalthalamica Gratioleti with the cortex of the lobus occipitalis are developed ad maximum.

By all means the best portion of the sub-section under consideration, however, is the extensive review of the results of physiological experiment upon the cerebral cortex. As might have been expected, the discussion here is masterously handled, and the reader is led in an orderly manner through the various researches dealing with electrical excitation, and extirpation of points and areas of the cortex. The cortical areas of the so-called motor zone, those for cutaneous and muscular sense, those for vision and those for hearing are successively considered, and the sub-section closes with some remarks upon the restitution of cortical functions after cerebral defect, together with brief reference to the doctrines of "association centres" in the brain as formulated by Flechsig.

The general pathology of the central nervous system is next taken up. The author's classification of cerebral diseases is given on p. 227. He divides them into four groups: 1. General or partial dynamic disturbances (including the "functional diseases"). 2. Diffuse and localized diseases of the brain and its membranes of circulatory and inflammatory nature. 3. System-diseases, and, 4. Focal diseases of the brain and its membranes. Only those of the fourth category are considered in the volume before us (and of those practically only the diseases dependent upon arterial alterations), the other diseases of the cerebrum being dealt with by various writers in other portions of Nothnagel's System.

The paragraphs upon pathological alterations in the ganglion-cells, like those dealing with the normal histological appearances of these structures, will scarcely prove satisfactory to those whose interest and research are directed chiefly toward such problems. Still, the principal points are mentioned, and it is worthy of note that the author includes certain important conditions not infrequently met with which are too often overlooked by investigators who are entirely under the spell of methylene blue and soap.

One turns naturally with much expectation of thoroughness to the chapter on secondary degeneration, and in so doing does not meet with disappointment. This portion of the book is admirable. The origin, course and significance of the degenerative process are fully described. Not only is the secondary degeneration in the sense of Waller properly valued, but the cellulipetal degeneration following injury to an axone, and the alterations in the cell-body of the neurone due to such causes are clearly described. The secondary alterations resulting from the application of the method of von Gudden receive especial attention. The finer histological changes are illustrated by drawings.

The atrophy of the II. Order, or so-called tertiary atrophy, for example such as affects the diencephalo-telencephalic neurones extending from the corpus geniculatum laterale to the lobus occipitalis after enucleation of an eye, or that which, after defect of the lobus parietalis, concerns the neurones, the cell-bodies of which are situated in the nuclei funiculi gracilis et cuneati, is clearly distinguished from ordinary secondary degeneration; the process is here so sharply characterized that there can no longer be any excuse for the confusion which has unfortunately marred many contributions to the literature of this subject in the past. Von Monakow, at the conclusion of the pages dealing with the degenerations, cites some of the more important examples of secondary degeneration in the brain of man and the higher animals after extensive cerebral lesion, especially those

which are of value from a clinical standpoint or of particular interest for general pathology.

To the clinician the portion of the book, amounting to something more than 100 pages, dealing with the clinical characters of organic diseases of the brain will prove unusually attractive. The author divides these into: (a) Phenomena of a general nature (including headache, vertigo, respiratory disturbances, alterations in the temperature and circulation, vomiting, and finally disturbances of a psychic nature); and (b) Focal phenomena (motor, ataxic, sensory and trophic). His profound anatomical and physiological knowledge here stands him in good stead, and the clinical symptoms of cerebral disease are, as far as present knowledge will permit, supplied with rational explanations.

The second principal part of the book treats of Cerebral Localization, a topic which, considering its complexity, the immense bibliography pertaining to it, and the uncertainty which still exists with regard to many fundamental points, is most satisfactorily handled. Everywhere one sees the hall-mark of a rich personal experience, and one cannot help wishing, after reading such an article, that medical publications in general could in some way be limited to those emanating from men who have some actual first-hand knowledge of the things of which they write.

The motor region, the parietal lobe, the visual area, the frontal gyri, are in turn dealt with, and the effects of gross and minute lesions described in detail. Of enormous value are the individual cases cited in which, following upon a thoroughly conducted clinical study during life, there has been a modern autopsy with not simply macroscopic examination of the central nervous organs, but also a thorough study of serial sections with the microscope. The time has gone past when the old-time slicing up of a brain at a post mortem examination can advance our knowledge of cerebral pathology. It is necessary now, in order to make progress, to determine exactly not only the actual nature and limits of cortical or ganglionic involvement in a given case, but also to establish definitely the extent of the disease in the white matter, and to decide how much of this is due to the direct encroachment of the primary pathological process and how much of it represents disease of a secondary nature. Von Monakow's laboratory, in Zürich, has been especially active in the sectioning of pathological human brains and has set an example to the world of what far-reaching results may be obtained from accurate studies of this kind. One brain fully described in this way is worth a hundred incomplete reports. Since the author, in this subdivision of his work, has not only carefully weighed his own results, but has instituted a comparison with those of others, the frequent consultation of these pages by both clinicians and pathologists may be predicted with confidence. It would require more space than that at our disposal to attempt an analysis of the various lesions dealt with here. Perhaps the most interesting section, though it seems invidious to select, is that dealing with disturbances in the central visual apparatus, under which heading hemianopsia, hemichromatopsia, cortical and mind-blindness, alexia and optic aphasia are discussed. The hypothesis of the Zürich neurologist with regard to the explanation of the behavior of the macula in hemianopsia is pleasing, but the anatomical proof has still to be brought.

The chapters dealing with the localization of disturbances of speech of cortical origin are worthy of being reprinted in the form of a separate monograph. It is in these that the scientific openness of mind of the author is perhaps most in evidence. The deliberate

presentation of objective, clinical and pathological facts to the exclusion as far as possible of the schemata and hypotheses which abound in works on aphasia is matter for gratitude. True, to the beginning medical student, one or another of the various schemes of aphasia is doubtless helpful, but to the man who attempts to gain a deeper knowledge of the subject, the method of inquiry which moulds its conceptions on realities is of far greater value. And after all, may not "students of the better sort" have some books written for them? There is reason to be thankful that there are certain books, like this of von Monakow's, that are something more than milk for babes! The clinical types of aphasia ordinarily met with are first described at some length, and afterwards the pathological anatomy of the disturbances of speech, and the finer localization within the speech region of the cortex are undertaken. This method of handling the material necessarily leads to considerable repetition, but with a subject which is even yet obscure, notwithstanding the strenuous efforts which have been made to illuminate it, rehearsals of known facts, especially when viewed from different visual angles, are not only permissible but desirable. If the reader were to carry away with him no more than a vivid impression of the possible variability of the clinical picture in aphasia and a consciousness of the inadequacy of all attempts thus far made to afford a satisfactory anatomical explanation for the various clinical types, the perusal of these pages would be well worth the while.

The section on localization includes also a discussion of lesions in the capsula interna, the basal ganglia, the cerebral peduncle, the corpora quadrigemina, the pons and the cerebrum, and closes with an exhaustive analysis of internal and external ophthalmoplegia.

The last two sections of the work deal with cerebral hemorrhages and with embolism and thrombosis of the cerebral arteries. The anatomy of the cerebral circulation is fully described, the descriptions being accompanied by excellent illustrations, many of which are in colors. The pathology and symptomatology of these affections are thoroughly gone into, not only in general, but with especial reference to lesions involving single arteries and their branches. Nor, as sometimes happens with writers well versed in pathology and diagnosis, have the sections on prognosis and therapy been neglected. The practical man, who wishes to know exactly what to do, not only in cerebral emergencies, but also in the often more puzzling after-care of a case, will find here explicit instructions for his guidance. A judiciously selected list of titles of original articles adds to the value of the various sub-divisions of the work.

The publishers have done their part well. The introduction of colors into the figures in the text makes decidedly for clearness. An occasional misprint meets the eye, for example "hereditären" on p. 220, and "Nourenencomplexen" on p. 225, but considering the size of the work and the rapidity with which it must have been written and put through the press, misprints are remarkably few in number.

On the whole, von Monakow's book represents a most valuable contribution to cerebral pathology, and can be warmly recommended to all who are interested in the healthy or diseased human brain.

LEWELLYS F. BARKER.

LEITFADEN DER PHYSIOLOGISCHEN PSYCHOLOGIE. In 15 Vorlesungen, von Prof. Dr. Th. Ziehen in Jena, mit 23 Abbildungen im Text. Vierte theilweise umgearbeitete Auflage. Gustav Fischer, Jena, 1898.

For the alienist this present volume is one of the most useful introductions to the study of psychological phenomena that we have

had the pleasure of seeing. The author allies himself most distinctly with the English psychologists as opposed to the teachings of Wundt in his "apperceptionslehre." That such a method of interpretation has great advantages from the practical point of view, the results of the teachings of Ziehen's earlier editions is abundant evidence. The psychology of Ziehen is distinctly not the old speculative introspective psychology of many writers, even modern: it is pure physiological psychology or, as he would seem to prefer the name, psycho-physics: a study of those psychical processes with especial reference to their physiological accompaniments: processes capable of being measured. And in his fifteen chapters he gives methods of determining quantitative and qualitative differences in psychical processes. He first discusses general considerations; the five following take up the primary types of sensation. Chapter 6, on sight, has been much enlarged and rewritten, constituting a distinctly new treatment of the subject. Chapter 8, on memory images and the formation of concepts, is mostly recast. Chapters 10, 11 and 12 treat of association of ideas and of attention, the subject of the ego and of memory. Chapter 13 deals with sundry psychophysical phenomena, somnambulism, hypnotism, etc., while chapters 14 and 15 discuss motor expression and activities, speech and the will. This present edition is certainly to be much commended.

JELLIFFE.

BOOKS RECEIVED.

"An American Text-Book of Gynæcology," edited by J. M. Baldy, M. D. W. B. Saunders, Philadelphia.

"A Clinical Text-Book of Medical Diagnosis," by Oswald Vierordt, M. D., and Francis H. Stuart, A. M., M. D. W. B. Saunders, Philadelphia.

"An American Text-Book of the Diseases of Children," by Louis Starr, M. D. W. B. Saunders, Philadelphia.

"A Text-Book of Materia Medica, Therapeutics and Pharmacology," by Geo. F. Butler, Ph. G., M. D. W. B. Saunders, Philadelphia, Pa.

"Clinical Lectures on Mental Diseases," by T. S. Clouston, M. D. Lea Bros. & Co., Philadelphia and New York.

"The Royal Road to Health," by Chas. A. Tyrrell, M.D.

"State of New York; State Commission in Lunacy; Ninth Annual Report."

"The Sexual Instinct and Its Morbid Manifestations," by Dr. B. Tarnowsky. Chas. Carrington, Paris.

"Vermischte Aufsätze," von P. J. Möbius. J. A. Barth, Leipzig.

"Arbeiten aus dem Gesamtgebiet der Psychiatrie und Neuro-pathologie," von R. Krafft-Ebing. J. A. Barth, Leipzig.

"Die Leitung der Electricität im lebenden Gewebe," von Dr. Fritz Frankenhäuser. A. Hirschwald, Berlin.

"Degenerados Criminosos," estado por Manoel Bernardo Calmon du Pin e Almeida. V. Oliveira & Co., Bahia.

"Untrodden Fields of Anthropology." Chas. Carrington, Paris.

"Les Actualités Médicales." Les États Neurasthéniques, formes cliniques, diagnostic, traitement, par Gilles de la Tourette. Baillière et Fils, Paris, 1898.

"Psychologie de l'instinct sexuel," par Dr. J. Roux. Baillière et Fils, Paris, 1898.

THE
Journal
OF
Nervous and Mental Disease

Original Articles.

A CASE OF SYRINGOMYELIA AND TWO CASES
OF TABES WITH TRUNK ANÆSTHESIA.*

BY HUGH T. PATRICK, M.D.,

Professor of Neurology in the Chicago Polyclinic; Associate Professor of Nervous Diseases, Northwestern University Medical School, etc.

CASE I. SYRINGOMYELIA. L. M., a Swede, aged 38 years, an iron moulder, was first seen December 12th, 1897, through the kindness of Dr. G. W. Johnson. He had smallpox when six years old, and a year or two later sustained a severe fall, which rendered him unconscious for four or five hours and bedfast for two or three weeks, but there seems to have been no fracture or serious local injury. Although unusually strong previous to the present affection, he had indulged to excess in alcohol and venery, and at the age of 27 or 28 contracted venereal sores of indeterminate character. This was repeated three years later, there being, so far as can be learned, on neither occasion any secondaries; nor is there any history of subsequent symptoms indicative of syphilis. On several occasions he had been severely chilled by exposure to cold drafts when greatly heated by his work, having fallen asleep where he lay down to rest.

In his opinion the present trouble began during the summer of 1889 or 1890. In the spring of one of these years (he is not certain which) he was made foreman, and in consequence had no occasion to do manual labor. In the autumn he resumed his former work, and then noticed that his back and

*Read at the twenty-fourth annual meeting of the American Neurological Association, May, 1898.

arms were not so strong as formerly. He had some difficulty in lifting and handling heavy objects. In all probability the disease of the cord had begun before this, for during the year preceding the first noticeable weakness he was never free from felons. These affected principally, but not exclusively, the right hand. Without apparent cause, and *without pain*, a finger would swell to about twice the natural size, feel clumsy for some days, finally discharge pus, and gradually heal. No sequestra came away, but on one occasion a good-sized slough, that looked like a tendon, separated, and when this was "pulled out by the roots" it hurt, but not excessively. In talking with the patient about it, I received the impression that this operation was very much less painful than it would have been in a normal person. What is still more conclusive is the fact that two or three years before the first motor symptoms appeared, he had a whitlow on the middle finger of the right hand, followed by diffuse cellulitis, for which three incisions were made, notably one on the dorsum of the hand about an inch and a half in length; and that these incisions were not very painful. About a year after he first noticed the diminished strength, he developed what was called erysipelas of the same hand and forearm. For this two free incisions were made and a large drainage tube drawn through. Neither the inflammation nor the operation occasioned suffering, although he cannot say that they were entirely devoid of pain.

Gradually the weakness of the back and arms increased, the left arm, he thinks, being rather worse than the right, and during the first year power in the legs also began to fail, as he can remember that sometimes after the day's work he was scarcely able to walk home, and found ascending steps particularly difficult. The ankles seemed weaker than the knees and hips. For two years after the beginning of the disability he was able to continue at his trade, with the exception of six weeks at about the end of the first year. At this time he awoke one morning to find the right arm and hand almost completely paralyzed. This seems to have been an ordinary pressure (sleep) paralysis, which allowed him to resume work in six weeks, although he thinks that the arm never fully regained its former usefulness. After this the right arm and hand were less efficient than the left. It should be remarked that three years previous to this sudden disability of the right arm, he had had a similar but less severe paralysis of the same arm, which I attribute to the same cause, and which kept him from work only two or three weeks. He was at the time of these attacks a steady and excessive drinker.

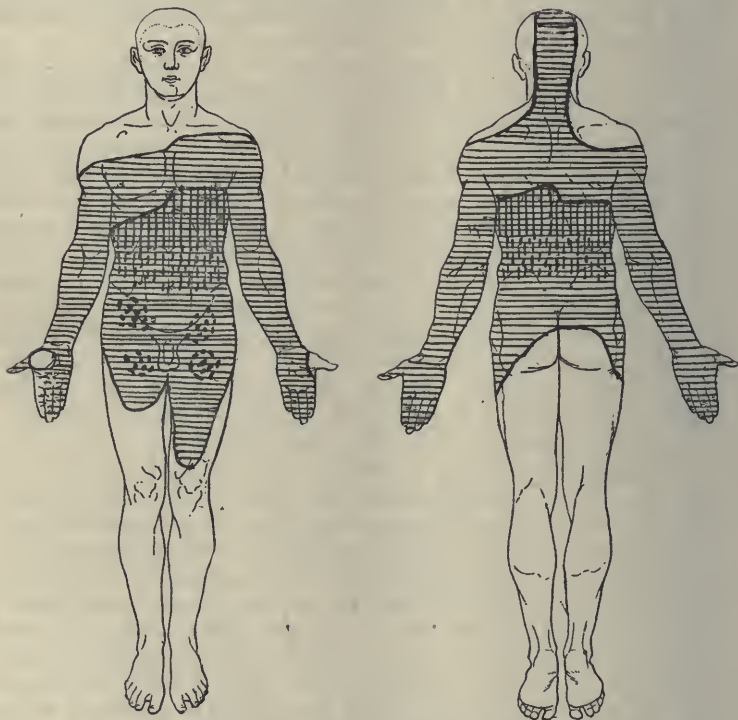
During the two years after the onset of the disease, when he was still occupied as a moulder, he constantly had blisters

on his hands, and was greatly puzzled because, as he expresses it, "his hands blistered so much easier than those of the other workmen." This was doubtless due in great part to the already existing analgesia. He was never conscious of burning himself; had not the warning of normal sensation, and consequently the blisters seemed to appear without adequate cause. I am inclined to think, however, that the skin of these patients may in reality blister with abnormal facility, as is the case in many other forms of paralysis. The tendency is seen in examination of the patient, for ordinary pin-pricks cause great weals to arise within a few minutes, and with a test tube of hot water I raised several blisters.

The power of the upper extremities has slowly decreased to the present time, the disability being much more pronounced about the shoulders than in the hands, but the patient affirms that the strength of the legs has improved somewhat during the last year. About a year and a half ago the hands, more especially the right, began to show vasomotor disturbance. They would frequently get red or dusky, and become somewhat swollen. The feet also became slightly enlarged, so that the patient was obliged to increase the number of his shoes from nine to ten. There has been no pain at any time, and no spasm or twitching, although he has noticed that after grasping an object for some time, the hand tends to cramp in the same position. Sexual power has been lost for four years, having begun to fail quite a year before its extinction. This seems relatively early for syringomyelia.

Examination reveals a sufficiently typical picture of this disease. Both shoulders droop, the right rather more than the left, the arms are pendulous, suggesting by their position and motion as the patient walks progressive muscular atrophy or dystrophy; the gait is typically spastic, but he "toes out" a little beyond the normal, and the feet are everted and flat, the right more so than the left. From the lower part of the neck down, all muscles seem to be more or less paretic, but very unequally so. The legs are more spastic than weak, the patient still being able to walk two or three miles at a slow rate, while the musculature of the upper arms and shoulder girdle is almost completely useless. Although he shrugs the shoulders with considerable power, the right trapezius above the shoulder, and the left one higher up, are decidedly atrophic and correspondingly weak. Indeed, at these parts they cannot be seen to contract at all. Both supraspinati and the right infraspinatus are wasted; the scapulæ seem too far from the spine, the borders are not parallel, and the patient is practically unable to approximate them, that is, to draw the shoulders well back. The deltoids are markedly atrophic, but although

the left is apparently more wasted than the right, he can raise the left arm almost to the horizontal for a moment, while abduction of the right is reduced to almost nil. Rotation is exceedingly poor, and worse on the right side. Biceps and triceps are wasted, flabby and very weak, the posterior muscles being rather weaker than the anterior, but even flexion is so feeble that feeding himself is a considerable task. Although the two sides in this location are much alike (circumference, R. $11\frac{1}{4}$, L. 11 inches), some movements at the shoulder and el-



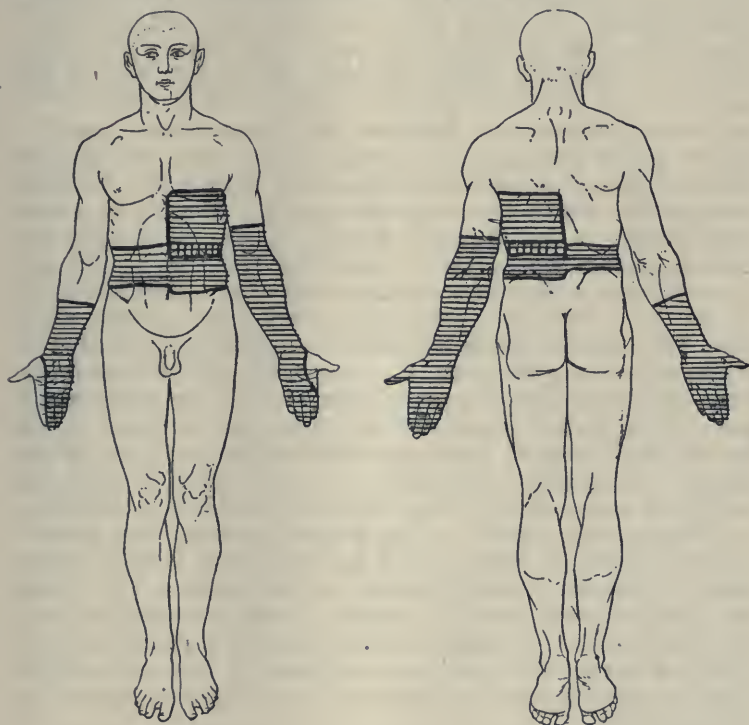
FIGS. I AND II. SYRINGOMYELIA.

Horizontal shading indicates analgesia. Perpendicular shading indicates tactile anæsthesia.

bow are better on one side and some on the other. The pectorals are not very strong, but could not be called atrophic. The forearm muscles are in a decidedly better condition than those of the upper arm. Atrophy is not apparent here and the grasp is fair, but the extensors on the left side are distinctly weaker than on the right, as in grasping strongly the hand flexes at the wrist, as it does in wrist-drop, although not to

an equal degree. Pronation and supination are quite defective, supination more so than pronation, and both worse on the right side. Spreading and approximating the fingers are feebly executed, more feebly on the left.

The cranial nerves are intact, including pupillary reactions and visual fields for white, blue, red and green. As in all cases of this disease, the sensory conditions are interesting. They are indicated in brief by the diagrams (Figures I to IV), which, however, must be supplemented by a few words.



FIGS. III AND IV. SYRINGOMYELIA. (Same case shown by Figs. I and II).

Horizontal shading indicates total loss of pain sense. Perpendicular shading indicates anæsthesia to firm touches.

First, the band of tactile anæsthesia about the trunk, although unmistakable, is not absolute. That is, to demonstrate and outline it, very light touches are necessary. The lighter the touch, the broader the anæsthetic band, and if quite firm touches are made with a camels-hair brush, no anæsthetic zone is evident. At no point on the body is there complete tactile anæsthesia. I may add that, as is the case with trunk

anæsthesia in tabes, the anæsthetic area is wider when examined from its middle toward the borders than when the latter are determined by approaching the zone of anæsthesia from above or below, where sensation is normal. Aside from this variation, the lower border of the anæsthesia is so uncertain that no exact limit can be determined. Second, although diminution of the pain sense is distinct quite to the limits indicated by Figures I and II, analgesia is not complete in all of this area. In Figures III and IV is shown the extent of complete analgesia. Third, sensation for both touch and pain is decidedly better on the right than on the left side. Fourth, the tongue of analgesia extending from the dorsal area to the vertex is not unique, although examples of it seem to be very rare. Gilles de la Tourette and Zaguelmann¹ report a case in which analgesia of the nape of the neck and back of the head existed as an isolated area, and Sölder² has recorded two cases in which the analgesia extended upward in a way quite similar to that shown in Figure II. In one of these the area gradually spread until it included the entire head except the face, a distribution that is not very exceptional. It should be added that, aside from variations already cited, there are spots of relatively small size, apparently located at random in the anæsthetic and, especially, in the analgesic areas, where sensation is more acute than in the surrounding parts. The patient seems to be rather more sensitive to the faradic brush than to other painful impressions. Loss of the thermic sense practically coincides in distribution with the analgesia, except that it seems to be less uniform in degree.

Plantar, cremasteric and abdominal reflexes are absent. The knee-jerks are exaggerated, as are also the Achilles jerks; there is incomplete ankle clonus on both sides. The wrist-tap (radius flexor reflex) is absent. From the regions indicated by dots in the diagrams, a lively reflex is excited by pin-pricks, although these are not at all painful, and occasionally this was noted in pricking the trunk about or below the level of the umbilicus.

The hands are "pudgy," thick, clumsy looking, a little on the "spade" order, inclined to be cold and cyanosed, but they are not deformed. The puffiness is resistant, and does not pit on pressure, and there is no enlargement of the bones. The feet present a similar appearance, and there is, besides, pes planus with, on the right side, so much eversion of the foot and protrusion of the inner bones of the tarsus as to suggest an arthropathy. There is no other joint trouble and no more lateral curvature of the spine than occurs in many normal

¹ *Nouv. Iconog. de la Salp.*, vol ii., p. 311.

² *Neurolog. Centralb.*, June 15th, 1898, p. 571.

persons. In the examination nothing has been discovered except the anæsthesia that would suggest leprosy. The patient says that often rotation of the head is accompanied with a grating in the neck, and this can be felt by the observer placing a hand upon the patient's head or neck. The feeling is something between that of crepitus and a click.

Changes in the electric reactions are not striking. All the atrophic muscles show diminished response to the faradic current, and in the atrophic portions of the trapezius the contraction is distinctly slow. These same portions (of the trapezius) show degeneration reaction to galvanism; that is, the response is very slow and the contraction long persisting. Other muscles respond by quick contractions.

CASE II. TABES. A. K., married, 52 years old, was first seen March 13th, 1898, through the kindness of Dr. E. R. Bennett. About 25 years ago he contracted a venereal sore, followed by a suppurating bubo and a number of enlarged glands in either groin. The inguinal and postcervical glands are now slightly enlarged, also the epitrochlear on the right side. There is no further evidence of syphilitic disease to be discovered either in the history or examination.

The first intimation of the present disease seems to date back about seven years, when, after dancing at a picnic, the legs felt tired out of all proportion to the exertion, and there was a sensation of tension or drawing about the calves and popliteal spaces. (This seems to be a frequent symptom of incipient tabes.) After this the same feeling was noticed when he was at work; as the patient expresses it, he felt as if he had walked a thousand miles. In the course of a year or so bladder symptoms appeared. With a sudden call to micturate there would be incapacity to start the stream promptly, and involuntary escape of a few drops of urine was not infrequent. A little later he began to have at longer or shorter intervals a sensation as if some one had suddenly gripped the left calf, and later still an occasional stinging pain in the leg or foot. Typical shooting pains have never been present. Five years ago, from no apparent cause, the left great toe became greatly swollen and congested; after a few days it "broke," giving exit to a little dark, bloody serum, and then rapidly became gangrenous. It was amputated by Dr. Bennett, and the wound healed promptly. There was some pain at the time of the acute swelling, but no particular attention was paid to the sensory conditions at that time. Shortly after the operation the left leg became enormously swollen, almost to the knee, but this swelling disappeared in a few days, and after several weeks he went back to work. It was then noticed that the left foot was everted, and that there was a

marked bony protrusion at the inner side of the instep. A few months after the operation a dusky swelling appeared on the stump, which discharged a dark serum for a short time, and then healed. At about this period the patient found that in putting on his trousers he had to steady himself by bracing his head against the wall. Nine months after the loss of the great toe the second toe on the other foot became swollen and dark, then turned black, and it was amputated three weeks after the first change. Union was prompt and perfect. By the time he had recovered from this incident control of the legs was too poor to allow resumption of his occupation. For about a year longer he got about with the aid of sticks and (later) crutches, but for the last three years has been confined to a wheeled chair, which he propels with the arms. He can now neither walk nor crawl, has had no sexual power for a year, and for the last two months there has been rectal incontinence when the bowels were loose.

Achilles-jerk, knee-jerk, wrist-tap and plantar reflex are absent, the abdominal reflexes are exaggerated. The left pupil is larger than the right, and there is reflex iridoplegia. Incoördination is very pronounced in the lower extremities, less marked but distinct in the upper extremities, and seems unusually prominent in the pelvo-femoral muscles. Sense of position is very much impaired and muscle tonus greatly diminished. The left trapezius muscle is atrophied in its upper (cervical) part, and the left arm is also somewhat wasted, being $1\frac{1}{2}$ cm. less in circumference than its fellow. There is entire absence of the pectoralis major on the right side. Of the latter muscle, there is a strong strand arising from about $2\frac{1}{2}$ inches of the clavicle; not a vestige below this. I consider this anomaly to be a congenital defect, as in the aplastic parts there is not the least trace of muscle, and yet the patient has never been conscious of any disability. As a carpenter he used plane and saw without inconvenience, and as a young man struck out from the shoulder like his companions. He says, too, that his mother in making clothes for him when a child remarked that he had a crooked chest.

For me, the interest in the case is almost confined to the sensory conditions and the spontaneous loss of the toes. Cases I and II taken together and considered in connection with some cases of syringomyelia, afford food for reflection and future comparison. A year ago I reported to this association³ a case of syringomyelia

³ Journal of Nervous and Mental Disease, October, 1897.

with trunk anæsthesia, and the preceding case (Case I.) constitutes an additional example of the same thing. The first one showed anæsthesia and analgesia more closely corresponding to the tegumentary sensory representation of the segments of the spinal cord than any reported up to that time, and, so far as I know, such a striking case has not been reported since. It showed the identical distribution often found in tabes, with the striking difference that the area of analgesia was large and that of tactile anæsthesia a narrower zone in the middle of the analgesic surface. The exact reverse ordinarily obtains in the trunk anæsthesia of tabes, as shown by Laehr,⁴ Bonnar⁵ and myself.⁶

The present case and the following one constitute striking exceptions to this rule. At least in the present state of our knowledge of this symptom in tabes, the topographical relation of analgesia and tactile anæsthesia shown by these two cases must be regarded as very unusual, and much more closely resembling that found in syringomyelia.

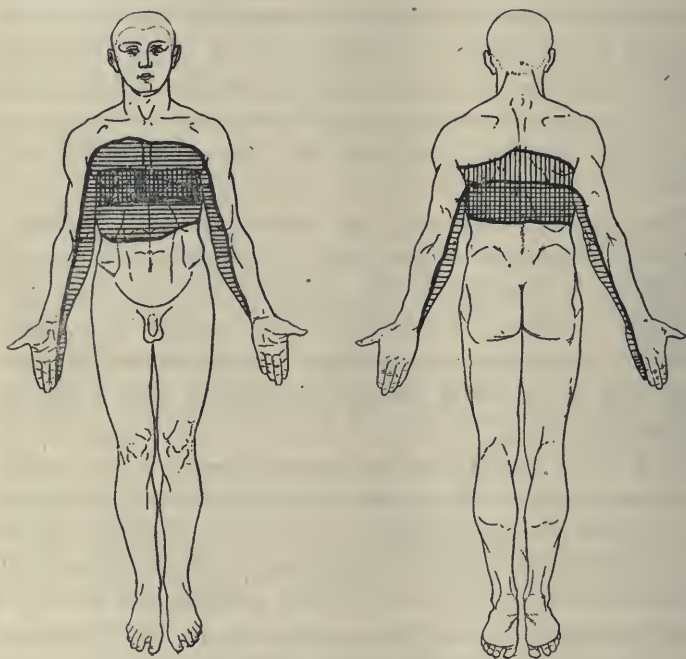
In A. K. (Case II.) the analgesia extends in front from the lower border of the second rib almost to the umbilicus, and includes the inner surface of the arms, stopping at the wrist on the left side, but covering the little finger of the right. (Figures V and VI). The analgesia on the arms, however, is not so marked as on the trunk, and the line bounding it is most indistinct; it is impossible to say exactly where it begins. The zone of tactile anæsthesia extends from just above the nipple to the xiphoid cartilage. Behind, the sensory condition is more like that prevailing in tabes. The tactile blunting extends higher, while its lower border practically coincides with that of the analgesia. Queerly enough, the latter extends on to the arms, and the former does not. Considering this anom-

⁴Arch. f. Psych., 1895, Bd. xxvii., Heft 3.

⁵New York Medical Record, May 22d, 1897.

⁶New York Medical Journal, February 6th, 1897.

alous distribution, it is proper to say that a number of examinations revealed the same condition. On the legs there is the usual dulling of both tactile and painful impressions, the former being the more in evidence. Sensory conduction is delayed, and impressions not at first painful often produce a burning sensation that is decidedly disagreeable.



FIGS. V AND VI. TABES.

Horizontal shading indicates analgesia. Perpendicular shading indicates tactile anaesthesia. The ordinary anaesthesia of the lower extremities is not indicated in the diagrams.

Atrophy of muscles about the neck and shoulders is not frequent in tabes, and the wasting of the upper part of the trapezius is of some interest, as being almost the exact counterpart of that found in the case of syringomyelia (L. M.). Probably still more exceptional is gangrene of the toes in this disease. Pitres⁷ has reported an

⁷ *Revue Neurol.*, 1893, p. 202.

instance, and so has Kornfeld.⁸ The case of the latter, however, was one of acute neuritis added to tabes. Joffroy and Achard⁹ have recorded a case of spontaneous gangrene in tabes, but the disease was already in the terminal stage; the patient had "pied-bot tabétique" with extreme tension of the skin due to the deformity, and died three days after the gangrene supervened. Besides, the gangrene in this case was not "in mass," but simply a gangrenous ulcer. Indeed, it seems strange that the occurrence is not more frequent, considering some of the other severe trophic accidents of tabes, such as the perforating buccal ulcer with bone exfoliation reported by Letulle and Lermoyez,¹⁰ Hudelo¹¹ and Leo Newmark,¹² and considering that there would seem to be principally differences of degree between perforating ulcer of the foot, whitlow with sequestrum and gangrene of the toes. The case that I report was once shown in a medical society as a probable example of Morvan's disease, and although I am not aware that the diagnosis between this disease and locomotor ataxia has given rise to difficulty, that between tabes and syringomyelia has been embarrassing to more than one observer.¹³ Even in the present instance, the sensory symptoms, the localized atrophy of the trapezius and the trophic lesion of the toes might, at a certain stage of the affection, have caused the diagnostician to hesitate.

CASE III. TABES. This case is sufficiently typical in every respect, excepting the sensory conditions on the trunk and arms. The patient is a man, 45 years old, who contracted a small venereal sore about 25 years ago, can recall no further symptoms indicative of syphilis, and first noticed slight incoördination of the legs seven years ago. Included in the history are pains in the lower extremities, transient ptosis and

⁸ *Semaine Méd.*, November 9th, 1892, p. 442, and *Wien med. Presse*, December 11th, 1892, p. 1,986.

⁹ *Arch. de méd. exper et d'anatomie path.*, 1889, No. 2, p. 24.

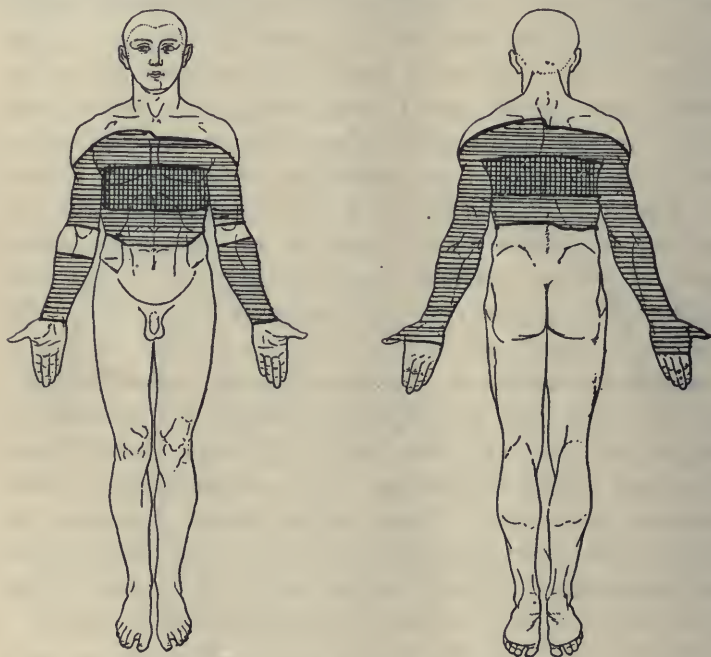
¹⁰ *Med Week*, 1894, p. 355.

¹¹ *Soc. franc. de derm. et de syph.*, May 13th, 1893.

¹² *Med. News*, January 26th, 1895.

¹³ *Parmentier: Nouvelle Iconographie de la Salpêtrière*, vol. iii., 1890, p. 213. *Bruns: Neurolog. Centralb.*, 1897, p. 511.

diplopia, impaired sexual power, and a moderate degree of cystic incompetence. At present there are small Argyll-Robertson pupils, loss of the knee-jerks, well marked ataxia of the lower extremities, slight incoördination of the upper extremities and complete analgesia of the ulnar trunk. On the legs the sensory blunting is that usually found in cases of tabes not far advanced; that is, tactile anæsthesia is very slight, analgesia very distinct, and both gradually decrease in degree



FIGS. VII AND VIII. TABES.

Horizontal shading indicates analgesia. Perpendicular shading indicates tactile anæsthesia. The anæsthesia on the lower extremities is not indicated in the diagrams.

from the feet upward. On the trunk and arms the distribution of impaired sensation is much like that already described as occurring in syringomyelia (Figures VII and VIII). Not only is the area of analgesia much more extensive than that of tactile anæsthesia extending from the trunk on to the arms (the tactile anæsthesia being limited to the former), but on the latter it presents the segmental distribution, with border at right angles to the limb, often seen in syringomyelia, but rare in tabes. In this case also the persistence of the peculiar sensory conditions has been confirmed by repeated examinations.

I regret to say that I have no explanation of the distribution and character of the sensory symptoms in these cases that is at all satisfactory to myself, nor have I even an opinion that is new. At the last meeting of the association Dr. Knapp¹⁴ presented an able paper covering similar cases, and I have now nothing to add to the several hypotheses and opinions then presented, but simply present these instances as a clinical contribution to a subject not very well understood.

DISCUSSION

Dr. P. C. Knapp said that a year ago he had read a paper before the association on the subject of sensory disturbances in cord lesions; he had been unable to throw much light on the subject then, and now he knew even less about it. Certainly, the study of sensory disturbances, especially in tabes, shows that they do not follow any definite course. Sometimes we get the well defined sensory disturbance of the so-called spinal distribution, at other times disturbances of the stocking, or glove, or sleeve type; sometimes such symptoms are absent entirely, or we may get one set of symptoms one week and another the next. In some cases we get analgesia; in others, analgesia with more or less anæsthesia or hyperæsthesia. As a rule, in tabes the analgesia is more extensive than the anæsthesia.

We certainly see cases of syringomyelia with analgesia, and considerably later in the course of the disease there is anæsthesia in a more limited area than the analgesia. In hysteria it is common to find analgesia on one side of the body without any anæsthesia.

In conclusion, Dr. Knapp said it was a question in his mind whether analgesia is the result of a slight disturbance of the sensory tract and anæsthesia is the result of a much greater disturbance, or whether we are dealing with two separate tracts, one for pain and one for tactile sensibility.

Dr. B. Sachs thought the view that the well known sensory symptoms of syringomyelia were almost pathognomonic of that disease could no longer be entertained. The speaker said he had observed two cases of Pott's paralysis, with dissociated sensory symptoms confined to the extremities; in two other cases dissociated sensation occurred first in the distribution of the trigeminal nerve. In one of these cases, seen about a

¹⁴ *Journal of Nervous and Mental Disease*, September, 1897.

year ago, there was absolute loss of pain and temperature sense in the distribution of the trigeminal nerve, while in other parts of the body sensation remained entirely normal. The speaker said he had no satisfactory explanation to offer for these unusual cases. In another class of cases he had observed symptoms which resemble those of syringomyelia, inasmuch as they have their origin in the cervical region of the cord, but they become absolutely stationary, and remain so for long periods—in some cases at least five years. An unusual feature of these cases is that the sensory symptoms are not as typical as they are in many cases of syringomyelia. Almost every form of sensation is somewhat involved, and there is not that sharp distinction between the analgesia and the anæsthesia that there is usually in syringomyelia.

Dr. Sachs said he wished to inquire whether cases of dissociated sensation within the distribution of the trigeminal nerve had been observed by any one else, and whether such cases developed the symptoms of syringomyelia later on.

Dr. Frank R. Fry said he had seen two cases of dissociated sensory disturbance of the fifth nerve. In neither of the two cases was any trophic disturbance noticed; there was merely a disturbance of sensation in the region of the fifth nerve. In one case this was more marked in the upper than in the lower division of the nerve. In the latter region it seemed impossible to produce pain.

Dr. Patrick, in closing, said he did not agree with the statement made by Dr. Knapp, if he referred to the impaired sensation on the trunk, that in tabes, as a rule, the analgesia is more extensive than the anæsthesia. On the trunk tactile anæsthesia is more extensive than the analgesia, and appears sooner, while in the lower extremities the reverse holds good. By some the view is held that the analgesia is due to a less pronounced involvement of the sensory tracts than that which causes anæsthesia, but this explanation is hardly adequate.

Dr. Patrick said that the dissociation of sensation on the extremities in several diseases was not very uncommon; for example, in Pott's paraplegia, multiple neuritis and tabes.

A CASE OF HUNTINGTON'S CHOREA WITH REMARKS UPON THE PROPRIETY OF NAMING THE DISEASE "DEMENTIA CHOREICA."¹

By FRANK K. HALLOCK, M.D.,

Cromwell, Conn.

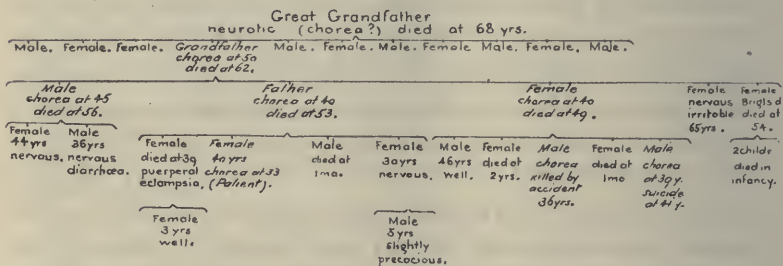
The histories of the majority of reported cases of Huntington's chorea begin rather abruptly with an account of the appearance of the choreiform movements and do not furnish data for the study of the earlier stages of the disease. The following case is, therefore, of interest chiefly because it shows clearly the course and order of development of the degenerative process, the dementia and chorea paralleling each other as the degeneration progresses. The case illustrates a well-recognized type of chronic progressive hereditary chorea, and viewing it from the psychological standpoint, one is strongly inclined to consider the chorea as of secondary importance to the underlying dementia, thereby warranting the descriptive and pathologically more correct name of "dementia choreica."

The patient is the wife of a college professor, native of Massachusetts, 40 years old, and has been under direct personal observation the past year. The accompanying table shows the hereditary history of the patient. It is not known whether the great-grandfather of the patient or his antecedents had chorea. The neuropathic tendencies, however, were transmitted through him. His wife was a very vigorous woman, and of their eleven children only one, it is believed, had the disease. This one, the fourth child, was the grandfather of the patient. He and his two sons used alcohol to excess during their lives. The mother of the patient came of better stock, but was probably never a very strong woman, and after the birth of the patient became a nervous invalid. Beyond what is stated in the diagram of descent, nothing is known of the rest of the family, except that a male cousin of the father was insane and confined

¹Read at the twenty-fourth annual meeting of the American Neurological Association, May, 1898.

in the asylum at Taunton, Mass. A glance at the family history shows the strong tendency to degeneration almost to the point of extermination. The reduced number of offspring, early deaths, and the earlier onset and somewhat shorter course of the chorea are significant facts.

The patient was never very strong, but up to the beginning of the disease she enjoyed fairly good health. No rheumatism, tuberculosis, or other special disease is reported in the family. She was married at twenty-one, and has had no children or miscarriages. In 1890, the thirty-second year of her age, the first symptom of the disease showed itself. This consisted of a mental change rather than any choreiform movement. There was, however, a single, isolated motion observed at this time, namely, a rythmical movement of the thumbs during church service. The mental change was the prominent feature and was manifested by a mild depression and lack of confidence in



Note. The names of those affected are in *Italics*.

herself. She did not complain, but seemed to be nervous and fearful of being left alone. If she went shopping without company she would become bewildered and lose her way home. In 1891 and '92 the same lack of confidence continued, and in addition the mental confusion and bewilderment increased. There was more difficulty in comprehension, and she would not talk with people. The motor symptoms accompanying this deepening of the mental condition consisted of no true choreiform movements, but of so-called "choking spells." While seated at the table she would suddenly throw her head back, gasp for breath, and then hurriedly reach for water to drink. Later on these throat spasms occurred in the night and seemed to be semi-hysterical in character.

In 1893 the mental condition of the patient changed. Instead of being afraid to leave home and reluctant to meet people, she now became full of confidence, wanted to travel and visit friends. Before, she was very subdued and anxious

to keep in the background; now she began to feel mildly exalted and pushed herself forward, emulating the acts of others. The "choking spells" at table and the thumb movements in church continued to be noticed with varying frequency. In 1894 the evidence of mental weakness became more noticeable. In ordinary sewing she could not learn to do the simplest things. Her memory began to fail.

In 1895 the first decided choreiform movements appeared. The "choking spells" grew less and were replaced by staggering movements which were first noticed in entering and leaving street cars or carriages. Difficulty in holding objects was experienced, knife, fork and other articles being repeatedly dropped. The menstruation at this time became irregular and remained so a year and a half. Greater mental confusion and failure of memory were observed. In 1896 the movements continued to increase in the extent of muscular involvement. When at rest, e. g., in church, she was quiet, with the exception of occasional arm or leg movements and a rhythmic motion of the head which consisted of looking up at the clergyman and then down to the pew. In walking the irregularity of movement became so great that she appeared to be intoxicated. Disturbance of speech and irregularity in handwriting were noticed. In 1897, her thirty-ninth year, she first came under observation.

Examination.—The patient is a medium sized woman, and, with the exception of the chorea and a rather blank facial expression, presents quite a normal appearance. There are no physical stigmata of degeneration. The heart, lungs and kidneys are normal and her general condition is excellent. There is no evidence of atheroma. The muscular development is uniformly well marked. The menstrual flow is regular, but for the last six months has been scanty. Vision, pupils and fundus normal. Hearing, smell, taste and general sensibility correct. Muscular and temperature senses perfect. No tremor or true ataxia. Romberg symptom absent. Skin reflexes present. Tendon reflexes active, especially knee-jerk, which is slightly exaggerated. No ankle clonus. Electrical reactions normal.

Choreiform Movements.—All muscles of the body are involved, including eyeball, tongue, pharynx, larynx and diaphragm. Patient cannot hold the eyes steady in the presence of another. They shift and wander almost constantly, due in part to irregularity of movements of ocular muscles and in part to motions of head. The tongue is protruded straight and has a large muscular development. It is moved about in the mouth and between the lips very unevenly, causing the char-

acteristic disturbance in articulation, which is labored, hesitating, broken in word sounds and sentences, ending often in a mild explosive utterance. The irregularity of action of the laryngeal muscles and imperfect control of the air column augment the difficulty in articulation. Coughing, choking, and a variety of minor vocal sounds are of frequent occurrence. Swallowing is similarly uneven in its execution. The movements of the facial muscles cause every form of grimace. The trunk and extremities are subject to all possible movements, producing an undulating distortion of the body and limbs from their normal position. The gait is wobbling and uneven, each step varying in its excursion as to height and latitude.

The two sides of the body are equally affected and all movements are greatly intensified in the presence of another person. When lying down or moving about alone the chorea is very much less. All motion ceases during sleep. Volition has a brief effect in quieting the movements. Occasionally the patient is subject (possibly as the result of dreams, certainly from some centrally initiated impulse) to convulsive-like movements at night, in which the body is tossed from one side of the bed to the other, or repeatedly wheeled around in a circle. Spasms of choking and difficulty in breathing also occur at times during the night. The character of the movements differs from that of the convulsive tics and Sydenham's chorea in being slower and more comprehensive. The motions are easier and wave-like and resemble more closely athetosis, without, however, any tendency toward contraction or fixation of the muscle group at the completion of the movement.

Mental Symptoms.—The ordinary appearance, behavior and conversation are fairly normal. The disposition and temperament, with the exception of the depressed condition at the onset of the disease, has always been happy and contented. Closer examination reveals distinct and decided dementia which is manifested by loss and inaccuracy of memory, both for near and remote events; a child-like simplicity, interest and pleasure in little matters; a lack of appreciation of her condition both as regards the choreic movements and mental deficiencies; a failure of the power to reason or be taught the simplest things, mental and physical. Volition is diminished, but there is an impatient and impulsive desire to follow out any idea, reasonable or otherwise, which comes in her mind. She has no hallucinations or delusions. Her mental condition, in brief, is like that of a child. She is very happy to the point of a mildly exalted state. Buries herself reading and fussing over little details in her daily routine. Enjoys social life, but is equally content to be alone.

Treatment.—Arsenic, bromide, hyoscine, hyosciamus and silver nitrate in ordinary doses had no effect on the chorea. In larger, paralyzing doses some reduction of the movements was noticed, but the reaction after ceasing their use was worse than before. Rest, isolation and the hygienic regulation of the daily life gave the best result, producing a temporary improvement noticeable both in the physical and mental conditions. After the experimentation with drugs was over and she went on the rest treatment the patient gained twenty-three pounds.

The following psycho-physiological considerations based upon the symptoms presented by this patient will be of service in correctly interpreting the true nature of Huntington's chorea.

Conceiving the arc of sentient life, or physical manifestations, to consist of three parts, we have on one side the flow of afferent stimulations from the periphery to the coördinating centres of the central nervous system, and on the other side, the sending forth from these centres efferent impulses resulting in motor activity. The nerve centres, spinal and encephalic, occupy the middle point of the arc. Examination of the nerves of the special senses and the general sensibility shows that the peripheral stimuli are, in the main, normally received and transmitted. Likewise testing the motor apparatus reveals the fact that the motor nerves transmit the efferent impulses, such as they are, properly and that the muscles are in good order and respond correctly to these impulses.

The cause for the irregular and abnormal behavior of the muscles, therefore, does not lie in the muscles themselves nor in their innervation, but in the peculiar character of the impulses they receive. These impulses arise in the nerve centres, hence we may conclude that here is the true seat of the disorder. The nerve centres chiefly involved are those of the cortical and subcortical motor areas and, disregarding the variable and coarser anatomical findings, the essential lesion, as described by Oppen-

heim¹, Dana², Michell Clarke³, Facklam⁴ and Collins⁵, is of the nature of a chronic, parenchymatous encephalitis.

The symptoms of this case support this view of the pathology of the disease in two respects, one is the fact that direct psychical influences are the most potent causes of the choreic manifestations; any emotional excitement or stimulation of the mental faculties greatly exaggerating the movements. The other is the complete change of consciousness, or ego of the individual, and the signs of distinct mental deterioration manifested in the form of a progressive dementia.

With comparatively few exceptions the histories of reported cases state that the chorea was noticed first and the mental symptoms appeared later. It is natural that this should be so, because the chorea as a rule is the conspicuous and absorbing symptom which brings the patient to the notice of the physician. Added to this is the fact that the disorder is quite commonly thought of and treated as a neurosis rather than psychosis, and the mental aspect of the case is consequently not apt to be so thoroughly investigated. (Charcot⁶, Lannois⁷, Jolly⁸.)

The writer is of the opinion that if more care were taken in studying the life history of the patient it would be found in a large number of cases that at the time of onset, if not before, distinct changes in the mental condition were already present. Huber⁹ was one of the first to report a case showing clearly the signs of mental deterioration previous to the appearance of the chorea. Several other cases have been similarly reported and re-

¹ Arch. f. Psych., xxv., 3.

² Journal Nerv. and Ment. Dis., Sept., 1895.

³ Brain, xx., 77, 1897.

⁴ Arch. f. Psych., xxx., 1, 1897.

⁵ Boston Med. and Surg. Journal, Dec. 23, 1897.

⁶ Lecons du Mardi, Policlinique, I. and II., 1887 and 1888.

⁷ Rev. de méd., Aug., 1888. Le bulletin méd., Nov., 1894.

⁸ Neurol. Centralblatt, x., June, 1891.

⁹ Virchow's Archiv., cviii., 1 and 2.

cently Facklam¹⁰, in an extended review of the disease, states that five of his eight cases showed previous mental change.

A comparative study of the different kinds of motor disturbance in connection with the various lesions of the central nervous system would be of distinct value in broadening our conception of such a disease as Huntington's chorea. Such a study should induce, on the one hand, the neurologist to consider cases of tic, Friedreich's ataxia, paralysis agitans, athetosis, etc., more from the psychological standpoint than is at present done, and on the other hand, the alienist would be induced to attach more significance to the play of the motor symptoms in the insane.

To illustrate the importance of taking a comprehensive view of the disorders of motility I will cite a case of generalized tic, characterized by pronounced motor symptoms with, as commonly considered, almost no mental peculiarities. In support of the view that the convulsive tics are, in the main, a degenerative disorder, we will find that the majority of cases, even of mild type, show certain psychical defects or departures in mental action from the normal standard. Noting, for instance, that the body and limb movements are rapid and changeable we will discover that the mental functions are similarly affected. The patient is apt to be restless and impulsive in conduct, finding it difficult to keep the attention concentrated on one line of intellectual work. The memory is often capricious and there frequently exists a superficial conception of the affairs of life. The mind is hurried and irregular in action just as the muscular movements. In fact the movements are almost exactly paralleled by the mental habits. Pursuing the examination further will reveal other associate imperfections of the psychical state.

My purpose in alluding in this connection to such a

¹⁰ Loc. cit.

disease as convulsive tic is to bring out the fact, first, that motor activity is the outward expression of the state of the motor centres, and secondly, that these centres are intimately connected by association fibres with those of higher consciousness¹¹ and, therefore, there is good reason to believe that whenever we find distinct and permanent motor disturbance of cerebral origin we may also expect to meet a certain kind or grade of mental deficiency or impairment. This impairment will vary according to the degree and type of the affection. It is true that it need not necessarily be present at all, as it is possible to conceive that the motor centres alone functionate imperfectly without involvement of the higher centre association fibres. The rule is, however, as Arndt¹² has clearly stated, that we will generally find some mental deficiency if we study carefully the habits, characteristics and inner life of the individual. Ordinary clinic or private visits, unless very frequent, often fail to reveal the facts of this kind. One must almost live with his patient to fully appreciate the psychical side of many of the so-called neuroses. Hence, in spite of the criticism, and citation of a case to the contrary, of so thorough a student of chorea as Sinkler¹³, we are forced by psychological reasons to believe that Diller¹⁴ is right in asserting that chorea long continued is almost invariably connected with mental abnormalities.

In the case of tic we may suppose the motor centres to be chiefly affected with slight involvement of the adjoining or association centres. In ordinary chronic dementia these adjoining centres of ideation, etc., are deeply affected, while the motor centres themselves are comparatively perfect in function, except as they are brought in

¹¹ Flechsig (L. F. Barker): "Sense Areas and Association Centres." *Journal Nerv. and Ment. Dis.*, xxiv., 6.

¹² *Arch. f. Psych.*, I., 3, p. 509.

¹³ *N. Y. Med. Record*, March, 1892.

¹⁴ *Am. Journal Med. Sc.*, April, 1890.

play secondarily, as is frequently seen in the rhythmic movements or habitual body posturing of such patients. Tic, paralysis agitans and athetosis manifestly cannot be considered psychoses, if we abide by the definition of the term, but Huntington's chorea, if we place it in the category with dementia paralytica and dementia senilis, and view it as an affection of the combined motor and association centres, may unquestionably be classed as a psychosis.

In determining whether the term "dementia" is applicable to Huntington's chorea it will be noticed that the point of view makes a difference in our conception. The neurologist chiefly interested in the motor symptoms is apt to underrate the picture of psychical decay before him, while with the student of insanity, if he has given the subject thought, there is far less difficulty in appreciating the unmistakable features of a true dementia with the disorder of motility occupying a position of secondary importance. Phelps¹⁵ makes an excellent plea in this latter direction, and a further witness on the side of the alienist's conception is the reviewer of Osler's "Treatise on Chorea" in the *Journal of Mental Science*, April, 1895, in which he raises the question: Why not a general chorea, as well as a general paralysis of the insane? It is interesting to note in this connection that Golgi¹⁶ pointed out, in 1874, the similarity in pathological lesions between chronic hereditary chorea and progressive paralysis. Since then this comparison has been made by several others. (Hoffmann¹⁷, Phelps¹⁸, Bondurant¹⁹.)

Setting aside for the moment the question of the propriety of the designation, dementia, we need be in no doubt as to the prognosis, course, futility of treatment and termination of Huntington's chorea. Given the

¹⁵ *Journal Nerv. and Ment. Dis.*, Oct., 1892.

¹⁶ *Rivista Clinica*, 1874 (abs. *Jo. Ment. Sc.*, xxii., p. 322).

¹⁷ *Virchow's Archiv.*, cxi., 3, p. 513.

¹⁸ *Hosp. Bull.*, Second Minnesota, 1892.

¹⁹ *Journal Nerv. and Ment. Dis.*, Oct., 1896.

heredity and the characteristic chorea appearing in middle life, the prognosis can be stated with a surety fully equal to that of dementia paralytica and dementia senilis. The slowness of onset of the mental deterioration and the cases exhibiting melancholic or maniacal symptoms do not in the least preclude the prognosis, and so in reality a diagnosis of progressive dementia.

One of the chief difficulties in considering hereditary chorea a form of dementia lies in the variation in type which is noticed to exist among reported cases. Thus, the cases cited by Hoffmann²⁰ and by Chaufford²¹, of chronic hereditary chorea without psychic disturbance make one question the completeness of their observations; the patient of Sinkler²², 34 years old, choreic from birth, and normal mentally; the two cases of Bower²³, one 36 years old, choreic at 30, with previous history of alcoholism and paraplegic attack, the other, 32 years old, with chorea of three years standing, not hereditary, and no mental symptoms; these and such other manifestations of chronic progressive chorea as have been observed must be considered as exceptions to the general type under consideration. The special character of the degenerative process and its manner of attacking the neuron, the rate of its progress and the locality of the tracts and areas affected must explain in this, as in all similar diseases, such variations as the above. Until more recently the cases have not been seen, or for some other reason, European authors seem to have had more difficulty in recognizing such a distinct form of hereditary chorea as exists in this country. Certainly American writers have portrayed a type, such as this case represents, with singular uniformity, and it is

²⁰ Loc. cit.—Questioned by Herringham, *Brain*, xi., p. 416; also by Sinkler, *Journal Nerv. and Ment. Dis.*, xiv., p. 80.

²¹ *Le bulletin méd.*, April, 1896.

²² *Med. Record*, March, 1892.

²³ *Journal Nerv. and Ment. Dis.*, March, 1890.

only to cases resembling this class that the term dementia is strictly applicable.

It may be argued that the term dementia can be applied with equal right to epileptic as well as hereditary choreic conditions. Practically this is just what is done when the dementia is well-defined, but the term is not serviceable earlier in the disease, as it is in the case of Huntington's chorea, because the appearance, course and duration of the mental deterioration cannot be foretold with satisfactory definiteness. The variable frequency of the epileptic attacks prevents the accuracy of prognosis, such as is attainable when we are dealing with a non-intermittent, steadily progressive process of degeneration such as exists in general paralysis, dementia senilis and Huntington's chorea.

This cursory review of the extent to which the body movements may express the state of the motor and associated higher consciousness centres must arouse interest in the effect of inherited tendency to degeneration as an etiological factor in the disorders of motility. Thus, it will be noted that in such diseases as tic, paralysis agitans, athetosis, Friedreich's ataxia, Huntington's chorea and dementia senilis the decadence of the neuron is more slowly progressive and is marked by a disturbance rather than loss of the power of movement. The paralysis, if present, is relatively less prominent and does not become pronounced until later. In dementia paralytica and other diseases of the brain and cord which are secondary infections, or to some other positive intercurrent process, the progress of the affection is more rapid and there is a combination of both disorder and paralysis of motion, the latter appearing earlier and as a more distinct symptom than is the case when the neuron loses its normal functions as the result of a natural mal-development or premature degeneration.

In conclusion it may be stated that a comprehensive

view of Huntington's chorea of the type here reported based upon the clinical, pathological and psychological data at our command warrants the following assertions:— first, that it is a progressive, degenerative disease of the brain, fundamentally different from ordinary or Sydenham's chorea; secondly, that the chief physical manifestations of the disease are the choreiform movements which are of secondary importance, merely indicating the nature of the cerebral lesion; thirdly, that the character of the mental symptoms can best be described by the term dementia; and fourthly, if the above assertions can be verified, then the disorder should be classed with such diseases as dementia paralytica and dementia senilis.

It may seem a stretch of the imagination to compare the tremor and other motor disorders of senility to the choreiform movements, but if we conceive of a degenerative process thrust violently upon the individual in middle life, the resemblance between the two diseases is not so unlike. Pathologically this is certainly what occurs, and the view here maintained is well set forth by Dana²⁴ in speaking of the disease as a teratological defect.

The propriety, as well as the advisability, of applying the name "dementia choreica" to Huntington's chorea is of course open to question, but if the term proposed excites a broader conception of the disease than is at present taken and directs attention to the psychological significance of the disorders of motility it will have served its purpose.

DISCUSSION.

Dr. J. J. Putnam thought that the view presented was a broadening one to the mind. Whether or not we had to do in such processes with joint conditions was always open to doubt. One saw the occurrence of various physical stigmata side by side with physiological and psychological evidence of degeneration, and sometimes without them. The same was true with regard to these morbid processes, but when the collocation

²⁴ Journal Nerv. and Ment. Dis., Sept., 1895.

became a fairly definite one, it was highly important to recognize it. What appealed to him most, perhaps, was the statement regarding a logical connection between disorders of movement and the psychical defects. Possibly that gap was not so wide as one might suppose.

Dr. Sinkler said, with regard to the renaming of the disease, that he had always thought the name "Huntington's chorea" was undesirable, both on account of the fact that, in general, the naming of diseases after individuals is undesirable, and because Huntington was not the first writer to describe this peculiar form of hereditary chronic chorea. It had been described as long ago as 1841 in Dunglison's "Practice of Medicine" by Waters, and in 1863 by Lyon. The name which Dr. Hallock proposed was, perhaps, also undesirable, because it led to a risk of confusion with chorea insaniens. He thought also that the writer made too sweeping an assertion when he said that all chorea of long standing leads to mental disorder. He quite agreed with him, however, that in the hereditary type, mental symptoms sooner or later develop without fail, but it is the rule that the mental symptoms develop long after the choreic symptoms begin.

Dr. Joseph Collins said it seemed to him that there was a more important reason than that given by the last speaker why Huntington's chorea should not be rechristened, according to the suggestion in the paper, namely, that in ordinary Sydenham's chorea there is often slight dementia. In fact, in every case of chorea of any considerable intensity and duration there is always a slight grade of dementia, described in the books as hebetude, mental sluggishness and the like, but which is after all a slight dissociation of the components of the mind. Hence, he could not see the propriety in singling out Huntington's chorea and calling it dementia choreica. There might be some who did not call up the clinical picture of mental and motor disturbance when the eponymic nomenclature was employed, but they were certainly very few. If we are bound to change the name of this disease he preferred to wait until we can designate by a term that shall encompass the pathological progress, and, perhaps, also the distinctive clinical features.

With regard to the basic lesion of Huntington's chorea, he had so recently written upon the subject that he would only say that it seemed to him fairly well proven, considering the findings in his own case, and in those of Dana and Oppenheim, which were all parallel, that the disease is a form of chronic parenchymatous encephalitis.

Dr. C. K. Mills thought the paper of Dr. Hallock was a

suggestive one, and, on the whole, the name proposed was a good one. The objections which had been offered to it by the previous speakers would hardly hold on a careful study of the matter. Chorea insaniens is a term applied to a definite form of acute or subacute mental disorder, which is distinctly different from that found in Huntington's chorea, and while some mental disorder is frequently present in many cases of chorea, using the word dementia in the technical sense, he scarcely thought it could be said that even prolonged cases of Sydenham's chorea lead to true dementia. Other forms of disease besides Huntington's chorea may do this. The paper is another evidence of the increasing interest being taken in the integrating of the numerous facts which we now have regarding the nervous system. With regard to the diseases that Dr. Hallock would associate with Huntington's chorea, it seemed to him unwise to class the latter with dementia paralytica. In dementia paralytica there were certain pathological conditions which in most cases were due to acquired disease of a peculiar type, the indirect cause frequently being syphilis.

Dr. Hallock, in closing the discussion, said that he thought Dr. Mills had answered the chief objections raised by Drs. Sinkler and Collins against the use of the name. In his own mind, viewing the question of psychical degeneration from the teratological standpoint, he was inclined to divide the process into three general grades: First, into the degeneration occurring in early life, represented by the adolescent insanities and mental abnormalities; second, into the degeneration in middle life, such as Huntington's chorea; third, into that which occurs at the climacteric period or later in life, represented by dementia senilis. Certainly the type of dementia senilis, which is more purely psychic and not markedly arteriosclerotic, can be considered, in one sense at least, a teratological defect, only further removed in lifetime than is the case with Huntington's chorea.

252. ZUR FRAGE VON DEN LAEHMUNGERSCHINUNGEN BEI PASTEUR'SCHEN IMPFUNGEN (Paralytic Symptoms following Pasteur Injections). L. O. Darkschewitsch (Neurologisches Centralblatt, 17, 1898).

The author presents the histories with clinical notes of two cases of paralysis following Pasteur's treatment of hydrophobia. The main symptoms were those of paresis of the extremities with paræsthesia pain and ataxia. The electrical reactions were less pronounced and there was some atrophy of the small muscles of the hands in one of the cases. In the second case there was developed a double facial paralysis.

Both cases recovered, though the first case retained some paræsthetic symptoms with slight ataxia of the fingers of the left hand for over a year.

JELLIFFE.

THE BRUCE MICROTOME.*

By C. EUGENE RIGGS, A.M., M.D.

The Bruce microtome, designed by my friend, Prof. Alexander Bruce, of Edinburgh, and made by A. Frazer, of that city, is intended especially for cutting celloidin preparations under the surface of alcohol. Its construction is very simple. It consists of a heavy metal tank, 2 cm. deep, 20 cm. wide and 90 cm. long, inside measurements. A square middle section is 6 cm. deeper than the other portions of the tank. In the centre of the deep portion is the object-holder. Orientation is secured through a ball and socket joint, which may be clamped to any position. The object-holder is moved vertically by a large but delicate micrometer screw, moving in a heavy sleeve attached under the centre of the tank.

Automatic feeding is obtained by a system of levers put in motion by the knife-block, and acting on a ratchet-wheel on the lower extremity of the micrometer screw. The knife-block is 4 cm. in thickness and 25 cm. long. It moves in a V-shaped groove running the full length of the tank. The cutting stroke is given to the block by a weight and pulley attached to one end, and the return stroke by a treadle and pulley. Two bronze arms, which may be adjusted at any desired angle, are attached to the block and suspend the knife within the tank.

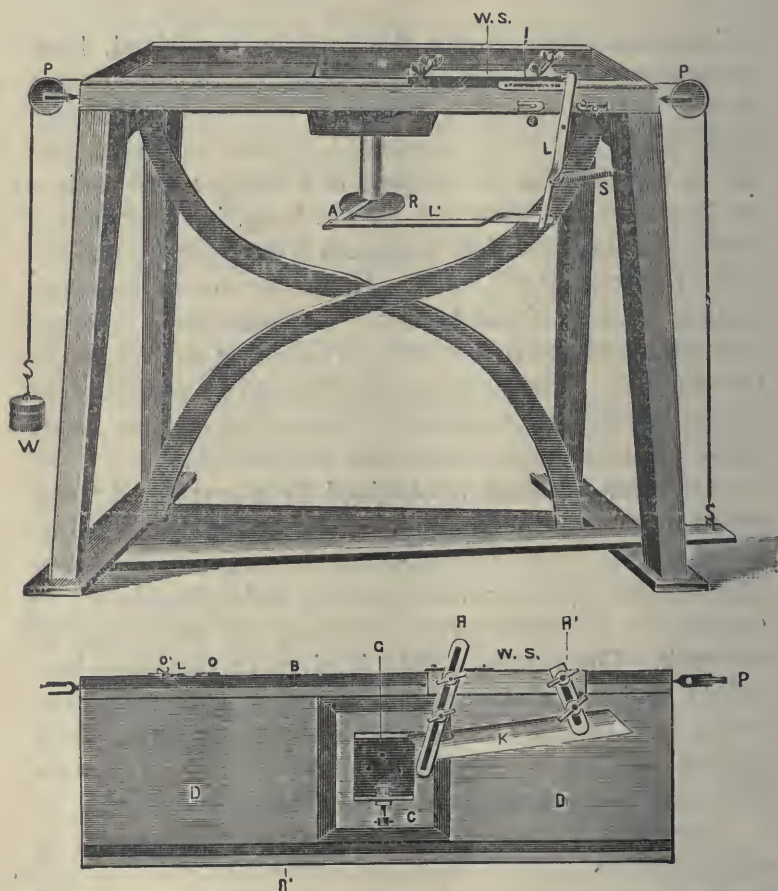
The knife is 5 cm. wide and has an effective cutting edge of 30 cm.

The whole apparatus is supported on four heavy metal legs.

In operation the knife is first adjusted; then the tank

*Read at the twenty-fourth annual meeting of the American Neurological Association, May, 1898.

is filled with 50 to 80 per cent. alcohol, sufficient to submerge the edge of the knife. From 1.5 to 2 litres of fluid are sufficient. When the block is in place and ready for cutting, the operator draws back the knife, which at the same time feeds the block upward, by a stroke of the



treadle. He removes his pressure from the treadle and the weight and pulley produce the cutting stroke. Both hands of the operator are free for handling the sections.

The good points about the instrument may be summed up as follows:

1. Its simplicity. Any intelligent person can understand it and operate it. One which has been in use two and a half years in my pathological laboratory has not once been in the least out of order.

2. Its accuracy. Exceedingly thin sections may be cut on it without "skipping." This is accomplished by the large size and rigidity of the parts preventing vibration, and by the fact that the entire block and the edge of the knife are constantly under alcohol.

3. Its capacity. While the object-holder is not sufficiently delicate in its adjustment for the finest embryological work, the largeness of the section is limited only by difficulties in embedding technique and by the width of the tank.

4. Its rapidity. The instrument is operated entirely by foot power, and both hands are free for removing the sections, which are very easily removed.

Several minor improvements might be made in the instrument at small additional expense, but even as it now stands it fulfills very perfectly all the requirements made on a celloidin microtome.

253. SUL TIPO PROGNEO NEI SANI DI MENTE, NEGLI ALIENATI E NEI CRIMINALI (On a Type of Prognathism in Normal, Insane and Criminal Individuals). G. Peli (Archio di Psichiatria, 19, 1898, p. 611).

The author discusses the type of prognathism in which the under jaw protrudes beyond the upper and gives a statistical study of much value. He shows that this condition is found in from 2 to 3 per cent. of normal individuals. In the insane the figures of various authors are given showing a large amount of variation. Richter gives 1 per cent. and Giuffrida-Ruggeri, 44 per cent., and others give intermediate percentages between these extremes. The author's own investigations covered some six hundred observations on the insane, one hundred criminal insane and two hundred criminals. His figures are about as follows. In the insane this condition was present in 28 per cent. of the men and 15 per cent. of the women; among the criminal insane, 33 per cent. among the men, and among criminals, 38 per cent. in men and 21 per cent. in women. Among the criminals this condition was more prevalent in those who had committed grave offenses, such as murder and robbery.

JELLIFFE.

A CLINICAL CONSIDERATION OF HERPES ZOSTER.¹

By LEONARD WEBER, M.D.,

Dr. Weber said that herpes zoster occurs usually on one side only, and that bilateral symmetrical involvement is very rare, although Eulenburg, Kaposi and others have observed such cases. Inasmuch as diseased conditions of the Gasserian ganglion and of the lumbar intervertebral ganglia in cases of herpes have been seen and described by Baerensprung, Kaposi and others, there can be no doubt that ganglionic affections, parenchymatous, hemorrhagic, etc., may be, and often are, the cause of shingles. Clinical observation has shown, however, that these are not the causes in every case, and that herpes may be produced by inflammation of peripheral nerves. Frequently, for instance, it does not follow the entire course of a nerve, but appears only along its most peripheral part. Again, it has been seen in the territory of nerves that were in a state of irritation or congestion through wounds, abscesses or tumors in their neighborhood. There are numerous reports by various authors of cases of herpes in which nodular perineuritis or interstitial and parenchymatous neuritis have been demonstrated in the affected nerve branches.

In Dr. Weber's own collection of twenty-three cases of herpes the largest number were intercostal; after these cervical and cervico-brachial were most numerous, then followed facial and abdominal, and finally, in a few cases the scalp or thigh was affected. The majority of the patients were not in good health when attacked;

¹Read at the twenty-fourth annual meeting of the American Neurological Association, May, 1898.

rheumatism, renal disease, uricacidæmia, spinal neurasthenia, spinal syphilis, being noted in them. The single case of herpes gangrenosus in the postauricular region of the occipitalis magnus branch of the third cervical nerve was in a tall, stout and vigorous man of forty-four years who was very nervous and excitable, but appeared to be in general good health.

Dr. Weber insisted upon scrupulous cleanliness in the local treatment of zona and dusted the part with bismuth powder or covered it with a 25 per cent. to 50 per cent. bismuth salve. He used ten to fifteen grain doses of quinine two or three times daily during a week or more for the severe neuralgia following herpes. Fowler's solution in rapidly increasing doses has not been of much service to him in the treatment of herpetic neuralgia. Ten to fifteen grains of iodide of potassium given three times daily with or without one-thirtieth grain strychnine, according to the presence or absence of cardiac weakness, have been valuable where the neuralgia was associated with œdematous swelling and induration of the parts. As long as the neuralgia stage lasts patients are very sensitive to even slight changes of temperature or motion of the affected parts. They must be kept very quiet in even temperature and carefully nursed. Prolonged exposure to cold and damp, severe muscular strain, acute and chronic infectious diseases and gastro-intestinal auto-infection play an important part in the causation of herpes. Dr. Weber observed a case of zona of the shoulders following scarlatina.

DISCUSSION.

Dr. J. J. Putnam did not think the argument advanced, that the whole nerve was not involved in cases of herpes zoster, was thoroughly sound. The vesicles indicated local invasion of bacteria from without at certain points, but they did not indicate to what extent innervation was impaired. In a case which he had seen, where the entire area of distribution of the supraorbital nerve was involved, the skin had become de-

pressed, thin and white, not only in the localities where the vesicles had occurred, but over the entire area, indicating a more widespread impairment of innervation than the distribution of the vesicles would have suggested.

Dr. Putnam was inclined to believe that the lesion in these cases was far back, to say the least, since in one case under his observation where the patient had suffered from repeated attacks of herpes zoster section of the supraorbital nerve was done as far back as possible, yet it did not relieve the pain.

Dr. W. G. Spiller thought that there could be little doubt that herpes is connected with disease of the nerves and ganglia, especially of the sensory fibres. He did not know of any case in which the vesicles were present with symptoms indicating involvement of the motor fibres alone. He had been able to observe a pathological condition of one of the spinal ganglia, which was probably the cause of herpes in a case studied in the service of Dr. Kovács, of Vienna. The patient had Pott's disease and suffered from an attack of zoster confined to a thoracic nerve of one side. At the necropsy the ganglion belonging to this nerve was found covered with miliary tubercles, while the ganglion just above, which had been removed by mistake, was apparently normal. Dr. Spiller thought that the evidence in favor of the existence of herpes from lesions confined to the spinal cord was not at all satisfactory.

Dr. W. M. Leszynsky said that in recent years he had come to regard herpes zoster as one of the symptoms of a peripheral neuritis, and had treated the patients accordingly with excellent results. For the purpose of relieving the acute pains, especially in the intercostal variety, he had found the Paquelin cautery remarkably efficacious. One application to the spine in the region of the posterior roots, corresponding to the affected nerves, was often sufficient to relieve the pain, and render special internal treatment unnecessary.

Dr. Wharton Sinkler agreed with Dr. Weber's view, that in herpes zoster the lesion was not always a peripheral one. It may be situated in the cord or spinal ganglia. The speaker mentioned the case of a woman of seventy, with rheumatic arthritis, who had an attack of herpes zoster along the anterior crural nerve; it was followed by a gangrenous condition of the part, and in spite of opiates, potassium iodide, arsenic, quinine and numerous other internal and local remedies, the woman died of exhaustion in three months.

Dr. C. L. Dana agreed with the view taken by Dr. Spiller that in most cases the trouble is in the nerve—probably in the sensory branch—and deeply seated. Practically, herpes zoster is mostly seen by the neurologist on account of the after-effects of the disease. It was not uncommon, he said, to see

zoster followed by severe forms of neuralgia affecting the intercostal, crural or facial nerves. He had seen a number of cases of *tic douloureux* following an attack of herpes zoster, and could also recall one case of very severe and obstinate paræsthesia affecting one of the intercostal nerves as the sequel of herpes zoster. The patient was given all sorts of drugs, underwent the "rest cure," and had a gynæcologist operate upon her, but all without avail. Finally she got well under toxic doses of strychnia.

Dr. Weber, in closing, said that as none of his cases had come to autopsy, he could not tell whether they were of local or central origin. He believed, however, that certainly one-half of the cases were of peripheral origin.

-
254. A STUDY OF THE LESIONS IN A CASE OF TRAUMA OF THE CERVICAL REGION OF THE SPINAL CORD SIMULATING SYRINGOMYELIA. James Hendrie Lloyd (*Brain*, 21, 1898, p. 21).

Lloyd here reports in extenso his post mortem findings in a case of traumatic affection of the cervical region of the cord, a full clinical history of which he had published in the *JOURNAL*, June, 1894. The case presented many features common to hemiplegia, yet that it was not a cerebral lesion would seem to have been indicated by the spinal deformity, the absence of paralysis of the face or tongue, sensory changes and the atrophy and fibrillation of the paralyzed muscles. It also showed the arrangement of the Brown-Sequard paralysis, paralysis of motion on one side and sensory changes on the other. In addition the peculiar dissociation symptoms and muscular atrophy of the shoulder and arm showed its relations to a syringomelic process. The patient died five years after the injury of an intercurrent disease.

The autopsy showed quite extensive flattening of the cord from about the fourth to seventh cervical segment, the seventh cervical segment being involved to the greatest extent, the whole cord here being flattened or ribbon like. The left half of the cord being greatly injured. The area of greatest involvement included the left antero-lateral column (pyramidal tract, cerebellar tract, and Gowers' tract), the anterior and posterior horns, the anterior portions of the posterior columns, and the gray and the white commissures. The detailed description cannot be here abstracted nor the excellent and full discussion of the physiology of the different fibre tracts. The author briefly recapitulates thus. "It may be said that tactile impressions pass directly up the posterior columns by way of the exogenous fibres of the same side, while painful and thermal impressions pass into the gray matter and through the cell bodies of the second order of neurons whose axis cylinders, in a large majority at least, pass across to the opposite side of the cord and up the lateral columns, especially in Gowers' tract."

JELLIFFE.

THE RESULTS OBTAINED BY THE OPERATION OF PARTIAL THYROIDECTOMY IN EIGHT CASES OF GRAVES' DISEASE.*

By J. ARTHUR BOOTH, M.D.

The histories of eight cases of Graves' disease with the results obtained by the removal of one lobe of the thyroid gland were reported. Five persons were cured by the operation, one was benefited, in one little improvement was noticed, and one died. The mortality from the operation is seven per cent.

DISCUSSION.

Dr. Theodore Diller, of Pittsburg, inquired whether any bad symptoms developed immediately or shortly after the operation in the successful cases reported by Dr. Booth.

Dr. J. J. Putnam, of Boston, said he had already reported two cases of thyroidectomy for Graves' disease, with death following in one instance and gradual improvement in the other. Since then he had had another case which resulted fatally after the removal of the cervical sympathetic. Although the general health of this patient was very poor, still the operation, apparently, precipitated his death, and the speaker thought that this case rather lessened the probability of the view that death occurring shortly after thyroidectomy is due to the absorption of thyroid products, as has been suggested.

Dr. Putnam said that a very favorable point in connection with the cases reported by Dr. Booth was that the operations were all done by the same surgeon. In all the more serious operations—those on the stomach, for example—the results are better in a series done by a specially trained surgeon than in a series of scattered cases done by different men.

Dr. W. M. Leszynsky, of New York, said he had had two cases of Graves' disease operated on during the past year. One of the patients had improved very much; the other died thirty-six hours after the operation. There was no kidney complication, and death was ascribed to acute thyroidism.

*Read at the twenty-fourth annual meeting of the American Neurological Association, May, 1898.

The case was a very pronounced one, and the patient was allowed to rest for nearly two weeks previous to the operation.

Dr. Booth, in reply to Dr. Diller, said that with the exception of a slight rise in temperature there were no alarming symptoms subsequent to the operation in any of his cases, except in the fatal case. He was not inclined to accept the theory that sudden death after these operations is due to the rapid absorption of the thyroid secretion; this, he thought, was hardly probable, as all the bleeding points are not only tied, but also cauterized, and the greatest care in the technique of the operation is exercised.

255. *THE PATHOLOGIC IMPULSE TO DRINK.* W. L. Howard, M.D. (Medicine, March, 1898).

Dipsomania is a symptom of defective inhibition. It is most common among those living at a high nervous pressure—physicians, litterateurs and business men. Exhibition of nervous energy always lessens inhibition. The cells of the cortex become exhausted by long-continued expenditure of energy; the individual resorts to alcohol to relieve his restlessness, the result of this cell exhaustion, and which prevents him from attending to his ever pressing duties. It is then that the defective inhibition is shown, and the uncontrollable impulse breaks the bounds of reason. The pathologic condition of the cells is probably analogous to the hypothetic pathology of hysteria. The protoplasm of the cortical cells becomes used up by continued work without the rest necessary for recuperation, and while in this state a small amount of alcohol rapidly cuts the higher centres off from the lower, causing a loss of inhibitory power. A continuance of these conditions results in such changes that each attack leaves the connection between the higher and lower centres less active, with a lessened amount of functional force in the cortical cells. Some cases of dipsomania can be directly traced to the absence of early education in not correcting uncontrollable impulses in childhood, but here we will find the child has inherited a richly neurotic soil. There is another class of dipsomaniacs, whose history shows early disturbance of cortical cells during their development periods. These are the cases which in infancy have been given alcohol in some form. Among other causes may be mentioned autointoxication. In considering prophylaxis and cure, it is important to bear in mind the somatic cycles by which many of our unconscious actions are governed. The long rhythms are habits of organic activity. The long rhythms in nutrition and heat regulations of the body are factors in augmenting the periodicity of dipsomania. Under pathologic conditions such as hypothetically exist in this psychic explosion, its intervals appear to be governed by the organic cycles, including the monthly rhythm of the female, and which in this sex, at this time, is often marked by slight attacks of dipsomania. Suggestion, with or without hypnosis, is of considerable value. Everything possible must be done to prevent the exhaustion of nerve force, and efforts made to store up reserve material. The physiologic rhythms must be watched, and when we see the approach of the ebb of these rhythms the patient must be carefully guarded.

FREEMAN.

CHLOROSIS AND RETINOPAPILLITIS.*

By H. M. BANNISTER, M.D.,

Member of the American Neurological Association; of the American Medical-Psychological Association and of the Chicago Academy of Medicine.

The following case is reported as possibly of interest in some respects, and because it is of a class that has been considered rare, though it is probable that such conditions are more frequent than has been generally supposed. Since commencing to write the account, I have heard of at least three other cases occurring within a short period in the practice of other physicians of my acquaintance, presenting more or less similar peculiarities, and giving rise to the same questions of diagnosis:

Miss J. M., aged 21, reported as previously always in good health, and with good family history, without tuberculous, cancerous or neurotic taint, so far as could be ascertained, was taken rather suddenly with a severe headache, on October 13th, 1896. This passed off in the course of the day, leaving her in her usual comfortable condition. The only precursors of the attack were a feeling for a week or two of becoming rather more readily fatigued on exertion than was usual, and a scantiness of the menstrual flow at the last regular period. She had always been normal in this respect, and had noticed the change, but she did not regard it as remarkable or consider herself as out of health, nor had anything been remarked on the part of her family as to her appearance or behavior. On the morning of the 14th the headache recurred with such severity, and was attended with such nausea and vomiting, that she took to her bed and called in Dr. G. P. Head, to whom I am indebted for the above facts, to prescribe for her. The headache was described by her as being more intense and more often on the right side, but only for a short period, and there was a point over the right parietal eminence that was decidedly tender to the touch. Any attempt to rise from the recumbent position was attended with severe nausea and vomiting of the cerebral type, though it did

*Read at the twenty-fourth annual meeting of the American Neurological Association, May, 1898.

not specially aggravate the headache. There were pronounced photophobia and an almost absolute anorexia and rejection of food by the stomach. The temperature was normal, or nearly so; at no time during this day or the following days did it rise more than a degree or two, and then only for short intervals, never much exceeding 100 and rarely rising above 99. The pulse was not specially accelerated, was not over 70, and otherwise not noticeably abnormal. The bowels were constipated and the urine scanty and high colored. Reflexes were normal.

The headache continued unintermittingly, as described, after the 15th, in spite of treatment with various anodynes, phenacetin, antipyrin, cannabis indica, bromides, ergot, etc., and became even more aggravated. Only morphine hypodermically was found effective, and this was administered in increasing doses, till one-third of a grain twice on the 17th and 18th and three times on the 19th was found necessary in order to obtain sleep and respite from the pain. The sleep was broken, and it was necessary by the 19th for the physician to administer one dose after midnight to carry the patient through till morning in tolerable comfort.

On the morning of October 20th I saw the patient with Dr. Head for the first time. She was, notwithstanding the little nourishment she had taken, not yet excessively reduced in flesh; her normal weight must, I judged, have been near one hundred and forty or forty-five pounds, though she was rather short in stature. She was pale and anæmic in appearance, lips and conjunctiva showing this decidedly; her tongue was covered with a thick, dirty appearing fur; her pupils were contracted, pulse was not over 50, and temperature was slightly subnormal. The bowels were said to be decidedly constipated; the urine was scanty, and was estimated at not over ten ounces in the twenty-four hours; its specific gravity was 1.030, its reaction acid, and it contained no albumin or sugar. The odor of the urine was rather peculiar, but this was, perhaps, attributable to some of the many drugs she had taken. She was very sensitive to any irritation, and the room had to be kept darkened, both on account of the photophobia and the nervousness produced by the light. Even the examination appeared to aggravate her headache, which, except when under the full influence of the morphine, was always more or less distressing; in fact, she claimed to be at no time entirely free from it. It was, as stated, worse as a rule on the right side, but the pain radiated down the back of the neck, and that region appeared stiff and tender. She complained much of roaring in her ears, especially the right, but there was no pronounced anæmic murmur. Her mind seemed perfectly clear;

her vision was at this time, she thought, perfectly unimpaired. There were some epigastric distress and tenderness, but none over the abdomen generally, and no motor symptoms other than those explainable by the pain and stiffness in the neck, and every movement of her limbs or change of position was distressing and dreaded on account of its liability to aggravate the headache. At no time had there been any delirious symptoms, convulsions, or signs of marked mental impairment; even the effects of a full hypodermic dose of one-third of a grain of morphine, with one-hundredth of a grain of atropine, were not noticeable, so far as causing any mental hebetude was concerned.

There was, and had been, no cough, and the lungs were healthy. The heart sounds were pure, and there was no pronounced anæmic murmur. There was a slight leucorrhœa.

It was thought advisable to have a more thorough examination of the urine made, and this was done on the 23d, when I saw the patient the second time with Dr. Head, her condition appearing practically unaltered in the interim. The urine was found, as on the former occasion, nearly normal, acid in reaction, had a specific gravity of 1.032, contained urea 3 per cent., considerable pus and epithelial cells, but no albumin or sugar. The blood examination showed hæmoglobin 45 per cent., red blood corpuscles 3,224,000. On this visit the patient, while in other respects unchanged, complained of a slight dimness of vision that had just become perceptible to her. The pupils were contracted as before and reacted normally; there were no anomalies of function in the ocular muscles. The vision was not tested other than to make sure that it was only very slightly impaired; she was able to distinguish objects fairly well in the darkened room, but said they were not as distinct as they had been. On account of the contracted pupils and the very nervous condition of the patient, the attempt to obtain a satisfactory view of the fundus on this occasion was unsuccessful.

On the strength of the blood findings, with the other symptoms, a provisional diagnosis of chlorosis was made, and the patient put upon arsenic and iron treatment in the form of Stearn's hæmoferrum, 3 grains every three hours, with 1-50 grain of arsenite of soda. Nourishment was given by the only method that had thus far been found successful, viz., a tablespoonful of Fairchild's panopepton with a little ice every two hours. Under this treatment, by the 26th of October, there appeared to be some improvement, the headache was less intense, and the morphine injections were reduced to three daily, the night dose being dispensed with. On the 27th, Dr. Head was able, by dilating the pupil, to get a view of the

fundus oculi, and found decided left optic neuritis, with a less advanced stage of the same in the right eye. The patient about this time also complained on one or two occasions of a temporary numbness of the right arm, with some impairment of motion in the same, but this did not come directly under Dr. Head's observation. Examination of the blood with the microscope during this time showed marked irregularity of outline of some of the blood disks.

I saw the patient for the third time, with Dr. Head, on October 30th, 1896. Her condition had so far improved that she was able to sleep nights without a second anodyne injection, and her headache was rather less severe during the day. She was still, however, taking three times daily one-third of a grain of morphine hypodermically, and her only nourishment was the panopepton. In other respects, her condition was the same as on the second visit, but the vision was, if anything, a little poorer, according to her statements. She seemed to see and recognize persons and things fairly well when close at hand, but complained of a certain vagueness and indistinctness in their outlines. As the pupils had been artificially dilated, I obtained a good view of the fundi. In the left eye the margin of the disk was altogether obliterated, and the only way to recognize its location was by a possibly darker shade and the convergence of the swollen, and tortuous vessels. In the right eye the condition was much less advanced, but there was pronounced optic neuritis.

The mental condition of the patient was the same on all occasions. She appeared bright and hopeful of recovery.

From this time on the improvement steadily continued, the headache became less intolerable, the stomach more tolerant of food, and on the 15th of November the optic neuritis had largely subsided; the vision was improving, according to the patient's own statement; she was able to be out and visit the doctor at his office. The morphine injections were discontinued, and Bland's pill substituted for the hæmoferrum, but through the month of November there remained a slight degree of headache and some nausea in the morning. Menstruation, which was scanty in October, beginning on the 5th, and was missed altogether in November, reappeared at the normal period on December 3d. On the 21st of this month she considered herself well; there was very little headache, if any; appetite and digestion were normal. The vision was considered normal, and examination showed an almost normal appearing disk in each eye. I did not myself see the patient professionally after October 30th, 1896, but can testify that her general appearance was that of perfect health. The iron and arsenic treatment was kept up till about the close of the

year, viz., Blaud's pill, 5 grains, arsen. strychn. 1-32 grain, arsen. soda 1-100 grain, t. d.

This observation is defective in several ways, in the lack of repeated determinations of hæmoglobin, of exact measurements of the visual defect, and in several other special points, where fullness and accuracy would have made it more valuable. As it is, however, it has, I think, a certain interest, as showing the severity of the symptoms that sometimes may accompany chlorosis, and the possible errors of diagnosis to which they may give rise. But for the suddenness of the onset of the headache and other symptoms, which suggested a doubt as to the existence of brain tumor, and led to the examination of the blood, that diagnosis would have seemed probably the correct one, and in fact I did not feel absolutely sure of its error till the success of the iron and the arsenic treatment had assured it. The case is almost unique, so far as I have been able to find, in the severity of the symptoms of headache and vomiting and the reported temporary motor disturbance of the right arm. As this last was not observed and tested by a physician, its importance cannot be altogether satisfactorily estimated, but the patient's statements are at least worthy of noting. Any hysterical element, I may say, was not at all prominent in the case, if it existed; the patient, in fact, appeared very little, if at all, hysterical in the usual sense of the word.

In this case the reduction of the hæmoglobin was not excessive at the time of examination, but the want of successive examinations affects the value of this fact. The subsequent poikilocytosis, which was very marked, would perhaps indicate a corresponding less percentage than was noted. The asymmetrical character of the optic neuritis is worth noting, though such cases are not uncommon, the right-sided occurrence of the headache for the most part, while the eye symptoms were most intense on the left, may also be worthy of mention. The rapid improvement under the iron and arsenic treatment is ac-

cording to precedents in such cases, and here it appears to have been instituted sufficiently early to insure perfect restoration of vision, a point that is insisted upon by Gowers.¹ In our case the visual disturbance was evidently only slight, as judged by the subjective symptoms narrated by the patient.

Optic disturbances in chlorosis have been noted for many years; the earliest reference I have found is that by Præel,² but they have been often ignored in the textbooks. Osler, for example, makes no mention of these symptoms, and there are other works equally deficient. Dieballa,³ in reviewing the literature, quotes Hayem (Du Sang, 1889), and Luzet (La Chlorose, 1892) as also not including optic neuritis in the symptoms of chlorosis, and speaks of the complication as one of the greatest rarities. That it is such I much doubt, but think rather that it is very commonly overlooked, sometimes possibly to the patient's serious detriment. De Schweinitz⁴ rather apologizes for introducing a case of this nature into a paper on "Monocular Optic Neuritis," and says it was introduced simply to call attention to the therapeutic measures desirable in such cases, thus apparently speaking of them as comparatively common occurrences. Ordinarily, it is probable that the symptoms are not severe, and the optic disorder may even progress till an incurable impairment of vision is produced. The following case, for the notes of which I am indebted to Dr. C. D. Westcott, is possibly the type of many others, some of which do not come under the care of any specialist or have the intraocular conditions recognized:

M. K., aged 27, bookkeeper and stenographer, came first under observation December 23d, 1895. She had for some

¹ British Medical Journal, 1880, ii., p. 780; also Med. Ophthalmoscopy.

² Bleichsucht mit Amaurose, Monatsschr. f. med. Augenh. u. Chirurg., Leipzig, 1840, iii., pp. 187-190.

³ Deutsche med. Wochenschr., 1896, No. 22.

⁴ Philadelphia Polyclinic, 1896, v., No. 50.

time complained of daily headaches and eye fatigue. She had always been nervous, and had had repeated attacks of nervous exhaustion. She thought her present health was good, with the exception of the above mentioned symptoms and fatigue from overwork. She was, however anæmic, and had amenorrhœa. Examination of the eyes showed externally only hyperæmia of the conjunctiva. Refraction test showed compound hyperopic astigmatism of moderate degree in both eyes. Vision with correction was normal, 20/20 in both eyes. Examination of the fundus showed marked hyperæmia of disk and retina of right eye. She was referred to the family physician for treatment of her general condition.

January 15th, 1896. Hyperæmia of disk still present, but stationary.

October 16th, 1897. She returned with reduced vision of right eye, 20/30; decided papillitis and general hyperæmia of retina and exudate in the macula. Fields for white were **normal**.

February 26th, 1898. Vision of left eye was normal; of right, 20/30; outline of disk was indistinct; no papillitis or retinal hyperæmia and no decided atrophy were noted; general condition was improved.

The details of treatment are not given in this account. We have here a case of anæmic, probably chlorotic, retinopapillitis, in which the patient was so far negligent of her condition as to let it progress till her vision was permanently somewhat impaired. Dr. Westcott had also notes of another similar instance, and they are suggestive, at least, of the possible greater frequency of chlorotic optic neuritis than is generally supposed to be the case.

Motor symptoms have not been generally reported, but the cerebral irritation that can cause optic neuritis can very possibly be enough to also give rise to local irritation or paralytic phenomena. Those in the case here reported are worthy, at least, of some consideration, and in Dieballa's patient there was strabismus. Severe sensory disturbances, headache, etc., rarely fail to occur, but it is only exceptionally that they are present to such an extent as in the case here reported.

The cause of the optic neuritis is, we may safely say, a cerebral irritation, the exact nature of which is, at best,

only a matter for conjecture. Lately certain French authors, Charrin,⁵ Etienne and Demange,⁶ have strongly advocated an autointoxication theory of the disorder, and this, while in accord with the trend of ideas at the present time, also affords a ready possible explanation of the special symptoms in these cases. It may be a too ready one, and the real facts may remain to be developed in an entirely different line; but just now it gives as plausible a theory as any at our command. If the menstrual function is really in any way an excretory one, as some have held, or if the internal secretion of the ovary may become perverted or insufficient in such a way as to poison the system and produce chlorosis, it is not going much further to presume that it may in special cases cause symptoms strongly resembling those of organic cerebral disease, and lead to embarrassing mistakes in diagnosis. The resemblances of this chlorotic optic neuritis, with its attending symptoms, to cases of brain tumor, have been already noted by Gowers and others, and have been illustrated by published cases. The one here offered is given as an especially striking example of this particular type.

⁵ Internat. Med. Congress, 1897.

⁶ Congrès Français de Med. Internat., Semaine Méd., April 23d, 1898.

256. THYROID EXTRACT FOR BACKWARD CHILDREN. Dukes (Brit. Med. Jour., i., 1898, p. 618).

The author reports very pleasing results from the administration of thyroid to a child described simply as "backward,"—from the rather meagre description, it would seem backward physically rather than mentally. She was well-formed and intelligent, but small; also very pale, in spite of administration of iron and arsenic. At the age of fifteen she measured 52½ inches instead of 58½ inches, weighed sixty-two pounds instead of eighty-eight pounds, and had the general appearance of a child of eight or nine. On a five-grain tablet of "thyroid extract" once a day, later increased to twice a day, she had lost her pallor, had begun to grow and was much more brisk and lively.

PATRICK.

BRAIN TUMOR SIMULATED BY ANÆMIA.*

BY HUGH T. PATRICK, M.D.,

Chicago.

As a supplement to Dr. Bannister's† more complete paper the following case may not be without interest:

B. M., a domestic, 21 years of age, was referred to me in February, 1895, by Dr. William H. Wilder, as she had "some symptoms of brain tumor." For about six weeks she had been suffering from constant and severe headache, more or less diffuse, but of greatest intensity in the left temporal and supraorbital regions, the pain often keeping her awake at night. Shortly after the beginning of the cephalalgia double vision made its appearance. This was not a simple blurring or confusion of objects, but distinct homonymous diplopia, due to paresis of the external rectus of the right eye. She gave a history of numerous momentary attacks of blindness, and complained of dizziness. There was intense double choked disk, with some small retinal hemorrhages, vision was reduced to 20/60 in either eye, and the visual fields were notably, though not markedly, contracted, as shown in Figures I. and II.¹

There was nothing of importance in the family history, except that the mother had died of cancer of the breast at the age of fifty. The patient had diphtheria when six years old, but after that had remained well and vigorous until two or three years before consulting Dr. Wilder, when she had begun to have about one yearly attack of moderate anæmia. When I saw her she pre-

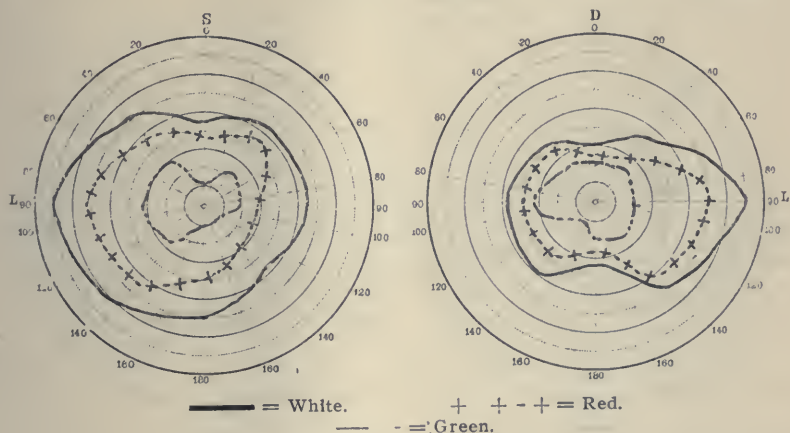
*Presented at the twenty-fourth annual meeting of the American Neurological Association, May, 1898.

† Journal of Nervous and Mental Disease, this issue.

¹ For Figures I. to IV. and for the eye findings I am indebted to Dr. Wilder.

sented all the signs and symptoms of anæmia except pallor of the face and lips. There was dyspnœa and palpitation on exertion, a feeling of languor, anorexia (especially for meat), and constipation. The vertigo, I found, was neither constant nor paroxysmal, but was caused by stooping, and more especially by rising from the stooping posture, and the attacks of transient amaurosis, which Dr. Wilder informs me have been emphasized by Hirshberg as a sign of brain tumor, were also confined to the moments of postural change. For several months there had been amenorrhœa. Examination revealed a soft blowing murmur over the base of the heart, a venous hum

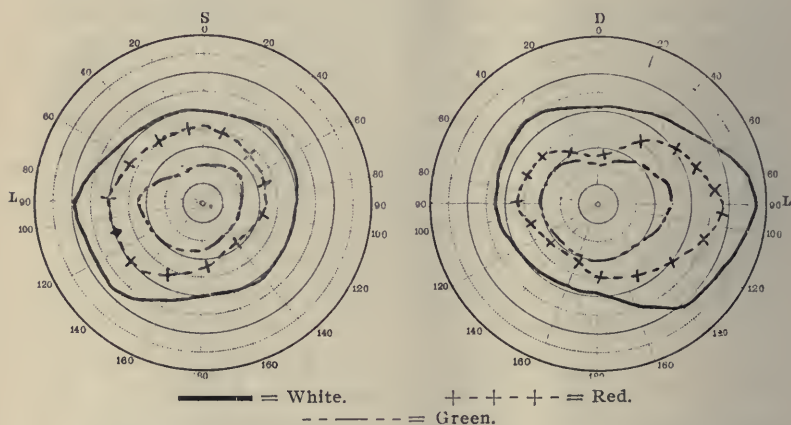
FIGS. I. AND II.



in the neck and 50 per cent. of the normal proportion of hæmoglobin. There was no evidence of tuberculosis or of any general or visceral disease. As no focal signs could be discovered, as the anæmia would account for all the symptoms except the paresis of the external rectus, and as this could generally be overcome wholly or in part by a strong effort, I made a diagnosis of anæmia only, excluding tumor for the time being. The patient was put on full doses of iron and arsenic, liberal diet and plenty of fresh air, and in a few days some slight improvement was perceptible in the amount of hæmoglobin, the ap-

pearance of the optic disks, the size of the visual fields and in the general symptoms. Progress for the better was constant, but rather slow. By the end of May (three months) the patient's condition was very satisfactory in every way, although she could not be said to be cured. The double vision had quite disappeared, but there was still some slight headache, and neither blood nor optic disks were quite normal. Examination at this time by Dr. Wilder showed vision to be 5/10 in either eye and the fields of vision decidedly improved, as shown in Figures III. and IV.; quite a material gain, it will be granted, but scarcely enough to prove my diagnosis beyond a peradventure.

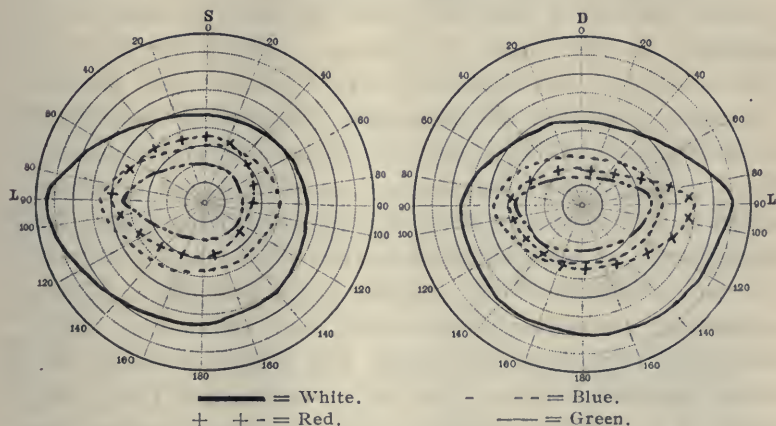
FIGS. III. AND IV.



During the next month she made some further improvement, and then passed from observation, until a recent letter brought her in to report—a little more than three years after the first examination. She says that she remained away because she considered herself cured, and has remained tolerably well ever since, except that every spring there was a slight return of the former trouble. On such occasions she took the same medicine (iron and arsenic) that had been prescribed for her, and always recovered within a reasonable time. The diplopia.

has not recurred, the headaches have never been so bad, and vision has never been so much reduced as at the time I treated her. At present she has no headache or dizziness, there is neither systolic murmur nor venous hum, and she has her full quota of hæmoglobin. In the absence of Dr. Wilder from the city, Dr. Robert Tilley was good enough to control my examination of the eyes, and reported that the left disk seemed rather hyperæmic, that the right "suggested a suspicion of beginning atrophy," and that the arteries in both seemed small. He also discovered one-half dioptre of astigmatism, and with a cylinder of this strength the patient's vision was normal.

FIGS. V. AND VI.



About a week later Dr. Wilder confirmed the results of this examination, adding that the left disk looked as if the swelling had not entirely subsided, and that along the vessels of the right fundus were distinct traces of connective tissue, evidently remnants of the previous choked disk. Evidence of this was also to be seen in a slight tortuosity of the vessels. I expected the doctor to find a weakness of the right external rectus, slight enough to be overcome in a state of health, but sufficiently pronounced to cause diplopia in the presence of exhaustion or extreme anæmia, but the Maddox rod and prisms re-

vealed only one degree of esophoria and normal ratio of abduction to adduction, although the dynamic condition of all the external ocular musculature was below par. The visual fields remain slightly contracted, as shown by Figures V. and VI.

In this connection it might be of interest to mention a case now under observation. A married woman, aged 32, had been for a period of three or four years to a great extent disabled by pelvic inflammation. This not only caused great suffering, but interfered with her appetite, general nutrition and disposition, so that she became thin and nervous, and spent a good deal of time in bed or on a couch. At length an operation relieved the pelvic lesions, and since that time she has had no symptoms referable to the uterus or adnexa. About the time of the operation, she began to suffer greatly from almost constant headache, to which was soon added occasional vomiting and left internal strabismus. After the operation, although the local conditions were all that could be desired, convalescence seemed delayed and the headache continued. A neurologist was consulted, who, I am informed, pronounced the trouble to be anæmia and nervousness. I saw the patient about four weeks later. She was very pale, thin and weak, but examination of the blood showed it to be practically normal, and there was nothing to indicate disease of thoracic or abdominal organs. The intense cephalalgia, pronounced paresis (almost paralysis) of the left abducens, typical choked disks, dizziness, a history of two abortive or atypical fits, slight difference in the knee-jerks and the patient's complaint of a weakness in one leg (although no difference could be detected on examination) pointed to the existence of a brain tumor; a diagnosis that seems to be confirmed by the subsequent course of the case, although only four weeks have elapsed since my first examination.

The case of anæmia was apparently one of tumor; the case of tumor apparently one of anæmia.

Periscope.

With the Assistance of the Following Collaborators:

CHAS. LEWIS ALLEN, M.D., Wash., D.C. R. K. MACALESTER, M.D., N.Y.
J. S. CHRISTISON, M.D., Chicago, Ill. J. K. MITCHELL, M.D., Phila., Pa.
A. FREEMAN, M.D., New York. H. PATRICK, M.D., Chicago, Ill.
S. E. JELLIFFE, M.D., New York. JOSEPH SAILER, M.D., Phila., Pa.
WM. C. KRAUSS, M.D., Buffalo, N.Y. HENRY L. SHIVELY, M.D., N.Y.
W. M. LESZYNSKY, M.D., New York. A. STERNE, M.D., Indianapolis.

CLINICAL NEUROLOGY.

257. DE L'ORGANOTHERAPIE OVARIENNE EN PARTICULIER DANS LE TRAITÉMENT DE LA CHLOROSE (Ovarian Organotherapy in Chlorosis). Werth (La Méd. Moderne, May 18, 1898, p. 318).

Werth, of Keil, is said to have been the first to employ (in 1896) ovarian extract in menstrual disorders, followed soon after by Mainzer, Moird, Landon, and later by numerous others.

The recent theories of the usefulness of this form of organotherapy are founded upon various views of the probable, or possible, part played by the internal secretions of the ovaries.

Spillman and Etienne, and Etienne and Demange, Von Noorden and others declare the usefulness of properly made preparations of ovarian substance, and say that no intolerance has been observed, even with prolonged use, and recommend an almost indefinitely continued dose after castration during troubles due to the menopause and in chlorosis, insisting upon the origin of the latter from disturbances or suppressions of the ovarian secretion.

MITCHELL.

258. RELATIONS ENTRE LES MALADIES DU SYSTÈME NERVEUX ET LA GLYCOSURIE (Relations between Diseases of the Nervous System and Glycosuria). L. Jumon (La Méd. Moderne, June 25, 1898, p. 405).

Numerous observations of late years have called attention to the relations between glycosuria, permanent or transitory, and disorders of the central nervous system, as well as in consequence of very various traumatisms, especially those of the cranial region.

It has been noted, too, that an alimentary glycosuria in persons in apparent good health may be followed by true diabetes. Von Jaksch, Strümpell and others have accordingly sought to establish a close connection between neurotic troubles and glycosuria, and have seen in the latter symptom a "neuropathic predisposition to diabetes." Van Jaksch goes so far as to believe the presence of glycosuria a fact of diagnostic importance in cases where traumatic neurosis is difficult of recognition.

Van Ordt, feeding a number of patients suffering from various nervous diseases, with an excess of glyucose and dextrose, found a large percentage (14 per cent.) presented glycosuria phenomena as a result. The experiment is hardly a fair one in view of the almost constant presence in neuropathic patients of gastric or intestinal disorder in some form. The experiment included 178 patients with widely different maladies, and the general conclusions to be drawn are not important. As might have been anticipated, glycosuria was not found constantly associated with any special diseases, but was a little more frequent with diseases of the encephalon. No doubt in many cases it was present as an expression of the general lowering of nutrition; but nevertheless it is true that its appearance and its fluctuations with the intensity of the nervous disease testify to a relation with pathological processes in the central nervous system.

In glycosuria consequent upon trauma the symptoms appear to be related rather to the nervous phenomena, to the traumatic neurosis, that is to say, than to the injury itself, and again individual predisposition must be taken into account, else we cannot explain why the same grave psychic syndromes should determine glycosuria in one case and not in another.

Glycosuria was noted in neurasthenia, hysteria and post-traumatic neurosis; it was not found in other neuroses, in true epilepsy, nor in general in spinal diseases not affecting the bulb.

W. Ebstein has examined the relations between epilepsy and diabetes from three points of view: 1, epilepsy as a result of diabetes; 2, diabetes as a result of epilepsy, and 3, the two maladies as a result of one cause.

The first is not a frequent case. In sixteen cases of diabetic coma Dreschfeld saw convulsions only once, and in eighty reported cases of coma but six cases of convulsions are noted, where albuminuria was not present to account for the seizures.

Jacoby considers diabetic epilepsy as an intermittent acetonæmic manifestation.

The second may be subdivided into two classes, according to whether there is simply a passing glycosuria after the attacks or a chronic diabetes. A passing glycosuria has been noticed by some former authors, but neither Ebstein nor Huppert has seen it, a result which agrees with Van Ordt.

True diabetes consequent upon epilepsy has never been noted except in Griesingers's case, which he attributed to an abuse of sugary food.

As to the third class, the causes may be general or local; hereditary predisposition and family habit may play a large part in both disorders, but the co-existence of the two diseases is certainly not common. Ebstein quotes one case. In two other cases with symptoms pointing to hemi-lateral cerebral lesions the patients suffered also with intermittent diabetes. Ebstein concluded that if glycosuria is not more frequently observed in epileptic patients, especially those suffering from the Jacksonian type of the disease, it is only because the urine is not frequently enough examined for sugar.

MITCHELL.

259. ZUR MULTIPLN HERDSKLEROSE (Concerning Multiple Sclerosis). M. Probst (Deutsche Zeitschrift für Nervenheilkunde, 12, 1897-1898, p. 446).

The writer reports a case which presented clinically the appearance of amyotrophic lateral sclerosis, but proved on microscopical

examination to be one of disseminated sclerosis. A man of fifty-four years had bulbar symptoms, with muscular atrophy and spastic paresis. Disturbance of deglutition and speech, atrophy of the tongue and lips, bilateral facial paralysis, atrophy of the small muscles of the hands, spastic paresis of the lower extremities, indicated involvement of the motor tracts. The classical symptoms of multiple sclerosis, intention tremor, scanning speech, ocular disturbance, etc., were not present, but the patient had vertigo and a somewhat slow reaction of the pupils. The lesions were confined to the medulla oblongata, pons and a portion of the corpora quadrigemina. Muscular atrophy is rarely seen in multiple sclerosis, but has been reported, and usually no change has been found in the ganglion cells of the anterior horns. Probst found in his case diminution in number and shrinkage of these cells in the upper part of the cervical cord. Secondary degeneration of the direct and crossed pyramidal tract was especially noteworthy, and extended from the proximal end of the pons into the lumbar cord. It was apparently due to a sclerotic area in the pons. Secondary degeneration has only been observed in a few cases of disseminated sclerosis. The columns of Goll were sclerotic from the upper thoracic region to the nuclei of these columns, and the intensity of the process was greatest in the upper part of the cord. The bulbar symptoms were explicable on account of the affection of the bulbar nuclei and nerves. Probst does not believe that this case was a combination of disseminated sclerosis and amyotrophic lateral sclerosis, because the vertigo was the first sign of the disease, because the muscular atrophy did not progress as rapidly as it usually does in amyotrophic lateral sclerosis, because the degeneration of the cells of the anterior horns was not as great as is usually seen in this affection, and because the secondary degeneration of the pyramidal tracts began in a sclerotic focus.

From a study of fifty-eight clinical cases of disseminated sclerosis, Probst finds that the sexes are about equally affected, that the disease is most common between twenty and thirty years of age, that exposure to cold and wet is the most common cause, and that infectious processes come next in etiological importance.

SPILLER.

260. EIN FALL VON ISOLIRTER FACIALIS-UND HYPOGLOSSUS-LAEMUNG NEBST PSYCHISCHER ALTERATION INFOLGE VON TYPHUS ABDOMINALIS
 • (A Case of Isolated Paralysis of the Facial and Hypoglossal Nerves with Psychical Alteration in Consequence of Typhoid Fever). A. Friedländer (Monatsschrift für Psychiatrie und Neurologie, 4, 1898, p. 110).

A boy, not hereditarily afflicted but not of normal psychical development, presented no unusual symptoms until he became fourteen or fifteen years old. He had been obedient until this time, but now became disobedient and excited, spoke much, ran from the house without any known motive, etc.; later he became more quiet and was able to work. He had a severe attack of typhoid fever, and during the convalescence he manifested symptoms of acute dementia and had paralysis of the right facial nerve. His behavior was that of a little child. He exhibited mind blindness, forced laughter and periodic attacks of intense anger, and made bad poetry; in short, he presented the symptom-complex of dementia præcox. The paralysis of the right facial nerve and, to some extent, of the right hypoglossal, was not believed to be peripheral, as the electrical reactions were normal; or cortical, as sensation was not affected. It was thought to be the result of a small area of softening or of a hemorrhage in the white

matter close to the surface of the lower portion of the anterior central gyrus. SPILLER.

261. CASUISTISCHE MITTHEILUNGEN AUS DEM GEBIETE DER MUSKELPATHOLOGIE (Clinical Communications Concerning the Pathology of the Muscles). R. Cassirer (Monatsschrift für Psychiatrie und Neurologie, 3, 1897, p. 491, and 4, 1898, p. 21).

Cassirer reports the clinical histories of several cases illustrating the difficulties of diagnosis in diseases causing muscular atrophy. The first case was in a young girl. The disease began with severe pain in the lower limbs and the patient became quite weak in these parts. Improvement was soon noted. About a year later the child had diphtheria and scarlatina with oedema. The weakness of the lower extremities increased and the motility of the arms diminished. No pain was experienced. After two years, during which time the improvement was gradual, the small foot muscles of the plantar surface, the quadriceps femoris, the ilio-psoas, and the adductors of the thigh, the deltoid, supraspinatus, infraspinatus, and triceps muscles were especially involved, while the remaining muscles of the thigh, pelvis, abdomen and back were little affected. The paralysis was not a degenerative one and pseudo-hypertrophy was not seen. The tendon reflexes corresponded in intensity to the degree of muscular atrophy. No objective or subjective disturbance of sensation was present.

The cause of this affection could not be neuritis or spinal lesion, chiefly on account of the absence of sensory changes during a long period, and the absence of a degenerative paralysis. The very slight fibrillary twitchings, the youthful age of the patient and the irregular and extensive atrophy, were not regarded as favoring the diagnosis of spinal muscular atrophy.

The variations in the symptoms and the atrophy of the shoulder muscles, with integrity of the muscles of the hand and forearm, were against the diagnosis of progressive neurotic muscular atrophy, while the acute onset of the process with pain, the many variations in the symptoms and the distribution of the muscular atrophy were not characteristics of progressive muscular dystrophy. The case was regarded as one of primary myopathy, resulting from polymyositis, and as not conforming to any of the known types of muscular atrophy.

Two cases of progressive neurotic muscular atrophy are described which closely resembled clinically the case just mentioned, but in these, qualitative electrical changes were noted. Although cases of muscular dystrophy with reaction of degeneration have been reported, they are very exceptional, and if such reaction occurs the diagnosis of muscular dystrophy can only be made when the muscular atrophy in its distribution is characteristic of this disease. Degenerative reaction Cassirer regarded as sufficient to exclude the possibility of progressive muscular dystrophy.

A case of acute anterior poliomyelitis is reported, in which later the signs of progressive muscular dystrophy were noted. No similar combination of the two diseases seems to have been recorded in literature. Cassirer says a connection may have existed between the two diseases, but that proof was not offered. SPILLER.

262. THE NEURASTHENIC SYMPTOMS OF GASTRO-INTESTINAL DISEASE. G. W. McCasky, M. D. (Med. Record, 54, 1898, p. 371).

There is one type of gastric disease in which for some reason the local sensory symptoms of the stomach disorder are almost entirely

wanting, and the local disease is more or less completely masked by the secondary toxæmic and neurasthenic phenomena, which cannot be successfully treated without removing the primary cause. More commonly, however, the gastric symptoms are not so latent. Intestinal symptoms are much more likely to be overlooked. The neurasthenic symptoms in intestinal disease vary greatly. General fatigue sensations are quite common. These may be mild and limited to a feeling of lassitude, with inordinate fatigue on slight exertion. In severer cases the various grades of neurasthenic pains are manifested sometimes in very aggravated forms. These pains may overshadow everything else and rack the patient's nervous system day and night, with disastrous results upon both the nerves and general nutrition. Incapacity for sustained mental effort may be shown in various degrees. This may be the result of vasomotor disturbances of the cerebral cortex, histochemical changes of the cortical cells, or the direct action of toxins. General sensory disturbances are very frequent. Paræsthesia and pruritis are the most commonly met forms. These paræsthesiæ generally occur in distinct episodes, lasting from a few days to a few weeks. Exhausting insomnia is among their effects, as the exacerbations are mostly nocturnal. Vertigo is very troublesome in some cases. It occurs in paroxysms during the height of digestion, or when the stomach is empty. In others it occurs whenever the patient shifts from the recumbent to the erect posture or vice versa. Muscular weakness is very frequent as a neurasthenic symptom, independently of the weakness expressive of the general debility from inanition present in aggravated cases of gastro-intestinal disease. The mental state tends toward depression. The digestive apparatus suffers like all other parts of the organism from lowered nerve function and thus it happens that in a large number of dyspeptics the gastro-enteric disease is primarily neurasthenic, but rapidly becomes more than this. The reason is that secretion and motility, the principal factors in primary digestion, are dependent upon nerve force. The vigilant germ is ever ready to seize upon the remnants of retarded digestion, producing gases and toxins, which by chemical and mechanical irritation still further impair secretion and motility, thus ever working in a vicious circle.

FREEMAN.

263. UEBER DAS "INTERMITTIRENDE HINKEN" UND ANDERE NERVOSE STÖRUNGEN IN FOLGE VON GEFÄSSERKRANKUNGEN (Concerning the Intermittent Lameness and Other Nervous Disturbances Resulting from Vascular Diseases). W. Erb (Deutsche Zeitschrift für Nervenheilkunde, 13, 1898, p. 1).

Erb reports a number of cases of this affection which was made well known under the title of intermittent claudication by the writings of Charcot. The literature is thoroughly reviewed. The condition in animals was recognized some years before Charcot reported a case in man.

Erb, with his assistants, has examined more than seven hundred cases, in order to determine the frequency of the absence of the pulse in the foot. He has found that at every age and in both sexes, when arteriosclerosis in a marked degree, cardiac trouble, or anomalies of the skin, do not exist, the pulsation of the foot arteries is present almost without exception, and its absence must be regarded as pathological.

Arteriosclerosis of the arteries of the foot with absence of every detectable pulsation may occur, without any indication of the intermittent lameness, nervous or vascular disturbances, or gangrene. In

such cases, however, the circulation must be sufficient for the needs of the part.

Intermittent lameness may exist without absence of the pulse in any of the arteries of the foot and without distinct evidence of arteriosclerosis. Arteriosclerosis of the deeper vessels or temporary vasomotor disturbance may be the cause of this lameness in such cases.

Purely mechanical, anatomical changes are not sufficient to explain the condition of intermittent lameness, but functional changes in the vessels are necessary.

The affection is usually of gradual development, and not infrequently at first is unilateral, but may be bilateral. Sensory disturbances, usually after walking or even during rest, are the first symptoms. These consist of a creeping or tickling sensation with a feeling of tension, coldness, sometimes of heat, more rarely pain in the feet and calves of the legs. Circulatory disturbances are generally early signs, and the feet are often blue and cold, especially when pendent or after walking. Motor weakness after use of the lower limbs, with spasm of the foot and calf muscles, manifests itself within a short time. The clinical picture is not always the same; sometimes the sensory disturbances, sometimes the vasomotor, sometimes the spasms are more pronounced, but the essential features of the disease are always present. The patient is entirely, or almost entirely, well during repose and when he begins to walk, but after walking a little distance the symptoms appear and he is obliged to rest a short time until his normal condition is restored. The arteries of the foot are found to be more or less diseased, and not infrequently pulsation is absent; in some cases the large arteries of the lower limbs are also sclerotic.

The intermittent lameness should be regarded as a danger signal of gangrene.

In the few anatomical investigations which have been made obliterating arteritis and chronic phlebitis have been found, and in some instances secondary changes in the nerves, muscles, skin, joints and bones.

The causes of intermittent lameness are those of arteriosclerosis, especially tobacco when used to excess, syphilis and extreme cold. Erb says there can be no doubt that excessive use of tobacco leads to arteriosclerosis, contracted kidneys, degeneration of the cardiac muscle, angina pectoris, etc.

The diagnosis usually is easy.

In the treatment all causes of arteriosclerosis should be removed, and energetic use of cold and hot water, mustard, vigorous massage, excessive movements of the lower limbs, tight bands, etc., are to be avoided. Articles of diet, such as strong coffee and tea, which affect the vasomotor system; drugs such as ergot and digitalis, which act on the vessels, must be forbidden. The feet and legs must be kept warm. Iodide of potassium should be given for the arteriosclerosis, and warm applications, and especially the galvanic (not the faradic) current, in the form of the galvanic footbath, should be employed to enlarge the vessels. Cardiac tonics which increase the blood pressure are desirable, and strophanthus is preferable to digitalis. Antipyrin or phenacetin may be used for the pain. Rest is of great importance, and every wound of the feet must be carefully attended to.

Erb suggests the name of *dysbasia intermittens angiosclerotica* for this affection.

SPILLER.

Under the above title, Biernacki (*Deutsche Zeits. f. Nerv.*, April 30, 1897) describes three cases of spinal cord disease that in course

and termination were exceedingly like acute myelitis, but in which a careful microscopic examination showed entire absence of inflammatory changes, the sole lesion being an endarteritis of the pial vessels. The duration of the disease from the appearance of distinct spinal symptoms to the fatal termination was from eight to twenty days, and, as stated, the clinical picture was that of acute myelitis; that is, quickly progressing paraplegia with sensory disturbance, paralysis of bladder and bowel and the rapid formation of massive decubitus.

The author is at great pains to demonstrate what has long been known to most neurologists; viz., that there is such a thing as softening of the cord from vascular occlusion, and that it is quite distinct from myelitis.

The obliterating endarteritis in the reported case was quite sufficient to produce all the symptoms noted, and the principal points of interest are: first, the very slight changes discovered in the nerve fibres and cells of the cord; second, the occurrence in one case of the sensory dissociation generally found in syringomyelia and almost peculiar to it (i. e., loss of pain and temperature sense with preservation of tactile sense); third, the peculiar relation of the knee-jerks to the other symptoms and the seat of the most advanced lesions; and fourth, lack of explanation of the rapid course of the disease, the arterial changes being essentially chronic in character. The last difficulty has apparently not occurred to the author, but would seem to demand elucidation. The almost normal appearance of the cord was probably due to the short course of the disease, sufficient time not having elapsed for the occurrence of chemical changes in the tissues that we know as disintegration and degeneration. Regarding the condition of sensation and the apparent inconstant behavior of the deep reflexes, it can only be said that there is much still to be learned concerning this, and cases of diffuse lesion such as those reported by Biernacki, are illy adapted for illumination on these subjects.

On the whole, we see no adequate reason why these three cases should constitute an excuse for adding a new term to neuropathology. Arterio-sclerosis of the spinal arteries, due to syphilis, senility and other causes, is well known, sufficiently comprehensive and sufficiently exact to embrace all such cases, but we do think that it should be more constantly in the mind of the practitioner than is at present the case.

PATRICK.

265. KLINISCHER BEITRAG ZUR LEHRE VON DER DYSTROPHIA MUSCULARIS PROGRESSIVA (A Clinical Contribution to Progressive Muscular Dystrophy). J. Hoffmann (Deutsche Zeitschrift für Nervenheilkunde, 12, 1897-1898, p. 418).

Hoffmann reports two cases in twin brothers which show that progressive muscular dystrophy may appear in the clinical picture of bulbar paralysis, contrary to the generally accepted opinion. The imperfect closure of the eyelids was noticed by the mother in both boys in early infancy, and this indicated that the disease was congenital or early acquired. Later, paralysis and atrophy of the facial muscles, atrophy of the tongue, paralysis of the soft palate—in one child paralysis of the muscles of mastication—were observed. The disease differed from bulbar paralysis in the involvement of the muscles of the forehead, and in the absence of fibrillary twitching and reaction of degeneration; and differed from the family form of infantile bulbar paralysis in the mask-like expression of the face, the tapir mouth, the lagophthalmos, the condition of the muscles elsewhere in the body, etc.

As differential points between the family form of infantile progressive bulbar paralysis (Fazio, Londe) and the bulbar form of muscular dystrophy, Hoffmann mentions the rapid progress of the former disease, as well as the fibrillary twitching, the reaction of degeneration, changes in other nerves (opticus), and the absence of the ordinary signs of dystrophy in the trunk and limbs. In some cases the diagnosis is impossible.

Between the congenital absence of the nuclei and the dystrophy of the facial muscles, the arrest of the process is a sign in favor of the former condition, especially when the process is unilateral and combined with abducens paralysis. In one of Duchenne's cases of muscular dystrophy, however, the atrophy of the muscles was confined to the face for thirty years. The occurrence of the atrophy in more than one member of the family and the tapir mouth are in favor of the diagnosis of dystrophy.

Hoffmann reports two cases which show that in muscular dystrophy the atrophy may begin in the peripheral parts of the limbs, and the condition may resemble that of progressive neurotic muscular atrophy.

Hoffmann shows in this paper that muscular dystrophy (1) may begin as bulbar paralysis and remain as such a long time; (2) that it may begin in the lower leg and forearm muscles, and (3) that external ophthalmoplegia may be one of its signs. All the voluntary muscles may be affected in muscular dystrophy, but no case has yet been reported in which the disease began in the small muscles of hand and foot. He is inclined to class the much-cited case of Oppenheim and Cassirer under the dystrophies, and not to regard it as a special form of disease.

SPILLER.

266. UEBER CENTRALE ERWEICHUNG DES RUECKENMARKES BEI MENINGITIS SYPHILITICA (On Central Softening of the Spinal Cord in Syphilitic Meningitis). Wullensoeber (Münchener Medicinische Wochenschrift, 45, 1898, p. 1,017).

A woman of 28 years, with a history of having had syphilis, presented the following train of symptoms: first, severe pains in the loins radiating to the front of the abdomen, next headache, then weakness and tonic contractions in the legs; patellar reflexes were lost, and she had intercurrent disturbances of vision, but no alteration of sensation for either touch, pain or temperature, and no ataxia. The trouble progressed, there was complete paralysis of the legs, with atrophy of the muscles, and sphincter paralysis, bed sores developed. The patient died about fifteen months after coming under observation. The autopsy showed syphilitic cerebro-spinal meningitis, with compression of the spinal cord, and the production of a central cavity extending from the midlumbar to the upper dorsal region of the cord. This cavity was most developed in the middle and lower dorsal region, where it occupied almost the whole of the usual site of the gray matter. No development of gliomatous tissue was to be found about the borders of the cavity. There was ascending degeneration in the cervical, and descending degeneration in the lower lumbar region. The arteries of both brain and cord showed characteristic syphilitic arteritis. On account of the absence of gliomatous tissue, the author thinks that syringomyelia can be excluded, and regards the case as one of necrosis of the spinal cord, due to syphilitic disease of the vessels. On searching the literature of the last twenty years he could find but four observations of cases in which syphilitic meningitis was accompanied by the formation of a cavity in the spinal cord.

ALLEN.

267. UEBER PUPILLENSTARRE IM HYSTERISCHEN ANFALLE NEBST WEITEREN BEMERKUNGEN ZUR SYMPTOMATOLOGIE UND DIFFERENTIALDIAGNOSE HYSTERISCHER UND EPILEPTISCHER ANFÄLLE (On Immobility of Pupils in Hysteria, with notes on the Symptomatology and Differential Diagnosis of Epileptic and Hysterical Attacks). J. P. Karplus (Jahrbücher für Psychiatrie und Neurologie, 17, 1898, p. 1).

Karplus here presents an extended series of observations with critical clinical histories of some eighteen cases of hysteria. Particular attention is paid to the condition of the pupils. In general he is opposed to the view that immobility of the pupils is of diagnostic value in the differentiation of hysteria and epilepsy, stating that in the attacks of hysteria major the pupils may be found to present exactly the same phenomena that are to be found in epileptic convulsions. In the consideration of hysteria without convulsions he shows that in these cases, three in number, the patients "lay as if dead" with closed lids, and in all of them there was immobility of the pupils to light.

Similar conditions were noted in the cases of hysteria with convulsive respiratory movements in which there was no loss of consciousness, also in cases with partial seizures and in still milder cases. The symptom has been found by him to be quite a common one in various hysterical conditions.

He further speaks of immobility of the pupils as a cortical phenomenon, believing that both dilatation and contraction have their representatives in the cortex, and that if for any reason the impulses from the cortex to the smooth muscle fibres of the iris are cut off, a condition of cramp exists in the iris musculature which is the basis of the phenomenon in question. Thus in a hysterical condition, a condition of cramp exists in the iris musculature similar to that found in the limbs whereby an analogous loss of the patellar reflex may be noted.

JELLIFFE.

268. ZUR LEHRE VOM RUECKENMARKSABSCESS. Concerning Abscess of the Spinal Cord. H. Schlesinger (Deutsche Zeitschrift für Nervenheilkunde, 10).

This case of abscess of the spinal cord reported by Schlesinger makes the tenth now on record. A woman of twenty-eight years was suddenly paralyzed in both lower extremities, and had complete loss of sensation and much pain in these parts. She had also a girdle feeling, rigidity of the muscles and tenderness on pressure of the vertebral column. Vesical paralysis and decubitus were noted. Death occurred about nine weeks after the beginning of the acute symptoms. At the autopsy an abscess, due to staphylococci, was found in the sacral and lumbar regions, extending into the lower thoracic portion of the cord. Peculiar foci were observed in the lower thoracic region, which were believed to be due to anæmic necrosis. A moderate degeneration was traced along the anterior fissure, through the thoracic into the cervical region, and was believed to represent the fibres of Marie's ascending sulcomarginal bundle.

SPILLER.

269. DERMATO-NEUROSES. (Savill. Clinical Journal, March 2d and 9th, 1898).

In two clinical lectures the author groups dermatoneuroses into "(a) those in which the lesion is situated in the course of a peripheral nerve (sensory or mixed); (b) those where the lesion is probably situated in the central nervous system (brain or cord); (c) those where the lesion is to be found at the end of the centrip-

etal or sensory nerve; and, finally (d), those in which the mischief is located in some part of the sympathetic nervous system."

In the first patient shown section of the median nerve had been done ten years before neuroses. The author divides the post-operation time into three periods. During the first two months there were what he calls paralytic symptoms. There was paralysis of the muscles and anesthesia of the skin supplied by the median nerve, as well as congestion, increased perspiration and glossy skin in its distribution. The first and second fingers became thinner. At the beginning of the third month began what he calls the irritation stage. The patient had neuralgic pains and tenderness along the course of the nerve, vesicles appeared on the knuckles and over the terminal joints of the index and second fingers, and the skin over the radial half of the palm was redder and more wrinkled than on the ulnar half. This condition lasted for six months, when the bulbous growth which had formed at the point of section was cut out and nerve-sutures done, after which the patient made a perfect recovery.

The second case was one of facial hemiatrophy, which came on after a severe bump on top of the head. The author considered it to be due to a paralytic lesion of the fifth nerve, because there was wasting and weakness of the muscles of mastication. Queerly enough, he does not state the condition of sensation, but the text reads as if it had been intact.

He next presented a case of severe facial neuralgia, with herpes on the forehead and grave changes in cornea and vitreous, as a case of "irritative lesion" of the same nerve. It is contended that an actual gross lesion of a sensory or mixed nerve produces skin changes, and that these differ with the nature of the lesion.

"A destructive lesion will produce glossy skin; and, also, if the lesion be severe and last long enough, extensive atrophy of the skin and its appendages. An irritative lesion in the same position will produce, though apparently in a shorter time, the opposite condition, viz., wrinkled or rough skin in the whole area of distribution; the vesicles will often appear in some parts of the area supplied by the irritated nerve."

As an illustration of tropho-neurosis from cerebral influence, the case of a young woman is cited who for three successive winters had suffered with pustular eruption on the hands. She was subject to hysterical fainting fits and attacks of migraine; but as she was employed at a large draper's where her hands were much exposed in handling the cloths, it may be fairly questioned if the case was one of hysterical skin affection.

Sclerodermic changes from syringomyelia were instanced among the tropho-neuroses of spinal cord diseases.

Two examples of skin affection supposed to be due to lesions of the ends of sensory nerves were shown. The first was a case of prurigo in an elderly man. The author believes that this disease is always caused by some disorder of the blood, generally due to some gastro-intestinal disturbance, and he finds a uniformly successful remedy in chloride of calcium. But the doses must be large, beginning with twenty grains and working up to forty-five grains, three times a day. The patient presented had obtained complete relief from thirty grains thrice daily, after meals.

As a reflex dermatoneurosis due to irritation of sensory nerves in a distant part, the author exhibited a case of facial chloasma in a young woman with endometritis and a small uterine fibroma.

Although only thirty-one years old, she had pronounced

wrinkles around eyes and mouth and the hands were harsh and wrinkled, all of which the doctor attributed to the condition of the womb. In a footnote he says that as the uterine functions gradually improved after curettement, the chloasma and wrinkles disappeared, to reappear four months later with a return of the menorrhagia.

Under tropho-neuroses, caused by disorder of the sympathetic nervous system, were classed Raynaud's disease, acroparæsthesia, giant urticaria, fugitive erythema and hyperidrosis. The vaso-motor cases have in common, paroxysmal appearances of the symptoms, symmetry of distribution, initial pallor followed by congestion, tendency of exposed parts to be affected and preponderance of women in the victims.

PATRICK.

270. THE LUMLEIAN LECTURES ON SOME PROBLEMS IN CONNECTION WITH APHASIA AND OTHER SPEECH DEFECTS. *Lancet* (April 3d, 10th and 24th and May 1st, 1897). Also ON A CASE OF AMNESIA OF 18 YEARS' DURATION WITH AUTOPSY. H. Charlton Bastian (Vol. 80 of the *Medico-Chirurgical Transactions*).

In his "Lumleian Lectures," the author begins with a consideration of the method of the storing up of word memories, and their reproduction as speech. There are three kinds of verbal memory. 1. "Auditory memory; the memory of the sound of words." 2. "Visual memory; the memory of the visual appearances (printed or written) of words." 3. "Kinæsthetic memory;" the latter being subdivided into "glosso-kinæsthetic" memories, i. e., "the memories of the different groups of sensory impressions, resulting from mere movements of the vocal organs during the utterance of words," and "cheiro-kinæsthetic" memories; i. e., "the memories of the different groups of sensory impressions, emanating from muscles, joints, and skin, during the act of writing individual letters and words." Since the centres for these different kinds of memories are not only in relation, each to its own afferent fibres, but are also closely connected with each other by commissural or associational fibres, the memory or recollection of a word in one, probably involves some simultaneous activity in one or more of the other centres. The centre for visual word memories is located in the angular gyrus and perhaps partly in the supramarginal lobule; that for auditory word memories in the posterior half or two-thirds of the upper temporal convolution. The author has previously stated his opinion that the motor centres of Ferrier and others "are really sensory centres of kinæsthetic type, by means of which movements are guided." The "glosso-kinæsthetic" centre is located in Broca's region. The "cheiro-kinæsthetic" centre cannot be so positively determined, but the author is inclined to follow Eauld, and to place it in the posterior part of the second frontal gyrus.

He suggests that the auditory and visual word centres are joined by a double set of commissural fibres, and that there is a particularly close connection between the auditory and "glosso-kinæsthetic" centres on the one hand, and between the visual and "cheiro-kinæsthetic" centres on the other. In the study of speech defects then, there are to be considered lesions, a, in the word centres themselves; b, in the different commissures by means of which these centres are connected; c, in the fibres connecting the two kinæsthetic word centres with their related motor centres in the bulb and cervical region of the cord; d, in these motor centres themselves. "Words are the symbols with which our thoughts are inextricably interwoven." Their revived "images" "may enter into thought pro-

cesses by more or less simultaneous renewal of activity in different regions of the cerebral cortex." The revival may be as the sound of spoken words, as the visual impressions of written or printed words, or as "the feelings of the muscular contractions concerned in the pronunciation of words." The two former are the more distinct and easily reproduced, the latter very vague.

There are two distinct and opposed views as to the method of word recall in ordinary thought. According to the first, words are revived "as faint excitations of the processes occurring in motor centres during the articulation of words." The second view is that words are revived mainly "as auditory ideas or images." This latter view the author strongly advocates, and gives his reasons for so doing very fully. Word recall may take place and doubtless does take place also as the revival of a visual memory, and in some persons the ability to recall words through the memory of their appearance when written or printed is very marked. In the great majority of people, however, the memory of the sound of spoken words seems that most easily revived. On this basis individuals may be classified as "visuals" and "auditives," according as the one faculty or the other predominates. "In ordinary persons, the four memories of words seem to be called into play in definite couples"—"the auditory and glosso-kinæsthetic revivals taking place during articulate speech, and the visual and cheiro-kinæsthetic revivals taking place during ordinary writing." The functional association between the auditory and glosso-kinæsthetic centres and that between visual and cheiro-kinæsthetic centres is extremely close. The word centres are in health excited to activity 1° by sensory impressions from without; 2° by association (i. e., by an impulse communicated from another centre during some act of perception or thought process); 3° by voluntary recall of past impressions. The author reviews the theories which have been proposed to account for the predominant influence of the left hemisphere upon speech without pronouncing for one or another, but insists upon the fact that the right hemisphere does not remain entirely uneducated, but receives to some extent visual and auditory speech impressions, and may possibly be able to some extent at least to take on a vicarious action, when the centres of the left side are injured. He strongly opposes the idea of a separate centre for concepts. Passing to the consideration of lesions producing speech defects, he confines himself to the study of those of the four word centres. The frequent association of aphasia with agraphia he ascribes to the proximity of the speech and writing centres, and of their afferent fibres to one another, combating the view that a lesion restricted to Broca's region will produce agraphia as well as aphasia. The question as to whether an isolated lesion of Broca's region will always produce verbal amnesia and alexia is answered negatively. While there is on record no case of pure agraphia, the author holds to his idea of a cheiro-kinæsthetic centre, suggesting that it occupies so limited an area that a lesion in that region is always large enough to produce paralysis of other movements of the hand and arm, at the same time as agraphia.

The division of aphasia into a motor and sensory form is not in accordance with his views. He prefers to limit the term aphasia to speech defects the result of lesions in Broca's region, using that of aphemia for those due to subcortical lesions, leaving those due to lesions of the visual and auditory centres to be grouped as amnesia, visual or auditory. He then proceeds to point out the most common combinations of symptoms of lesions in the various word centres, illustrating them by numerous case histories. A close study of these

shows surprising and at first sight contradictory results. While insisting throughout upon the superior importance for speech, in most persons, of the auditory centre, the author thinks that some cases of speech preservation where the auditory centre was destroyed, may be explained by the theory that the affected individuals were "strong visuals." In other apparently anomalous cases, extending over years, the original injury may have been less extensive than that found after death. In those cases in which power of speaking has been gradually regained, he thinks that the right auditory centre may have been by degrees educated to act through the commissural fibres upon the glosso-kinæsthetic centre of the left side. He explains the cases of word blindness without agraphia, by assuming that, the subjects being strong "auditives," the cheiro-kinæsthetic centre was influenced directly from the auditory centre. The cases of destruction of the centres in both hemispheres are so few and their histories so incomplete that the author does not attempt to explain their symptoms at any length. For a discussion of the forty-three cases mentioned, see the original. In his other paper the author gives a very complete history extending over eighteen years of a remarkable case of speech defect, in which, though the autopsy showed complete destruction of the angular and marginal gyri, of the superior and part of the middle temporal convolutions, and of part of the ascending frontal, second frontal, and ascending parietal convolutions on the left side, Broca's region being uninvolved, there was neither word deafness, word blindness, nor agraphia. Spontaneous speech was however very limited, and though the patient could read and understand what he read, and could copy with his left hand, he could neither write spontaneously, nor from dictation, nor could he read aloud.

ALLEN.

271. PARALYSIS OF THE SIXTH NERVE FOLLOWED BY DIPLEGIA: RECOVERY. Wood (Brit. Med. Jour., Apr. 3, 1897).

The author reports an interesting case, apparently of acute toxic or infectious origin, but thrombosis was not excluded.

A healthy lad of 18, of gouty heredity, gradually developed during two days, paralysis of the right sixth nerve and in the succeeding five days, left hemiplegia, including the lower part of the face. The orbicularis palpebrarum and the frontalis were not involved and sensation was normal except for a feeling of numbness on the paralyzed side. The hemiplegia rapidly improved and in two weeks had quite disappeared, but four days before this, distinct bulbar paralysis came on and in five days had become very marked, the tongue, lips, pharynx and probably larynx being affected. From this condition the patient also rapidly recovered and a month later was practically well excepting the abducens paralysis, a trace of which remained five months after the beginning of the trouble.

The author seems unwilling to make a diagnosis, but likens the case to an acute poliomyelitis of the medulla and pons—a comparison that seems to us to be eminently rational.

PATRICK.

272. THYROID CHLOROSIS. Dr. Capitan (The Medical Week, 5, 1897, p. 609).

Dr. Capitan, noting the well known fact that in chlorotic subjects the thyroid gland is frequently enlarged, states that Professor Hayem has found this to be the case in twenty-nine patients out of thirty-five under his observation. The goitre is usually very small and very soft, though sometimes it is pulsating, in which latter case

the general symptoms of mild Graves' disease are usually present. The author thinks that the chlorotic condition is dependent upon this small degree of exophthalmic goitre, and is due to one of the varieties of thyroid intoxication. He considers this further demonstrated by the fact that such cases have improved rapidly under the administration of a rather strong solution of iodine and iodide. Such patients treated exclusively by tablets of iodothylin lose the chlorotic and exophthalmic phenomena within a few weeks.

MITCHELL.

273. UEBER DIE HEILUNG ASEPTISCHER TRAUMATISCHER GEHIRNVERLETZUNGEN (Concerning the Healing of Aseptic Traumatic Cerebral Wounds). T. Tschistowitsch (Ziegler's Beiträge, vol. 23, No. 2).

The writer reviews carefully the literature on the regeneration of nerve tissue, and shows that the results obtained by the different investigators are contradictory. He himself performed a number of experiments on rabbits, dogs and pigeons. He experimented in three different ways, he thrust a cold or hot needle, or small tubes made of celloidin into the brain, or he excised pieces of cerebral tissue, always under antiseptic measures. The most important conclusions which he formed from his studies are, that after cerebral injury the restoration of tissue is almost entirely accomplished by proliferation of the connective tissue of the pia and vessels. The neuroglia plays an unimportant part, and forms merely a secondary sclerotic zone about the scar or foreign body, and this only in those cases in which the irritation from the wound is unusually great and of sufficiently long duration. In gradual destruction of the specific elements of cerebral tissue this glial sclerosis may possibly be more intense. This hyperplasia of the neuroglia is probably caused by special forms of irritation. He was not able to observe any regeneration of nerve cells, but is more guarded in his statements concerning the possibility of regeneration of nerve fibres. The capability of the ventricular ependyma to proliferate is very slight. The ependyma may cover a limited portion of the inner surface of the brain which has been deprived of these cells, but it has no part in the restoration of nerve cells or of cerebral tissue.

SPILLER.

274. ACROMÉGALIE CHEZ UN NEGRE AGÉ DE 14 ANS (Acromegaly in a Negro aged Fourteen Years). Valdès (La Presse Médicale, No. 78, 1897, p. 174).

Valdès (of Matanzas, Cuba) reports a case of acromegaly in a fourteen year old negro boy. The excellent reproductions of his photographs, and the outlines of the foot and hand of the patient as compared with those of healthy individuals, show that the disease was fully developed and characteristic. The patient suffered from headache and from muscular weakness, and besides enlargement of the hands and feet, presented an increase in size of the lower jaw and a cervico-dorsal kyphosis. He had no visual nor auditory disturbance.

ALLEN.

275. LA GHIANDOLA TIROIDE NEGLI ALIENATI (The Thyroid in Mental Affections). P. Amaldi (Revista Sperimentali di Freniatria, 23, 1897, p. 311).

In an extensive article of some forty pages, the author presents the results of a study of some 107 cases, 69 men and 38 women. In the men some 58 per cent. showed changes in the thyroids and in the women 53 per cent. of the thyroids were affected. In senile dementia and in pellagra the changes were more manifest. Colloidal and granular degenerative changes were the most common types of lesion found.

JELLIFFE.

Book Reviews.

LES MYÉLITES SYPHILITIKES, FORMES CLINIQUES, ET TRAITEMENT, PAR LE DR. GILLES DE LA TOURETTE, PROFESSEUR AGRÉGÉ A LA FACULTÉ DE MÉDECINE DE PARIS (Actualités médicales), J. B. Baillière et Fils, Paris, 1898.

Spinal syphilis is a subject of some importance, since in its clinical forms it closely resembles certain types of spinal disease which have a bad prognosis and which are not amenable to treatment, whereas in certain cases of spinal syphilis treatment is singularly efficacious. The author here has given in small compass an excellent picture of the various syphilitic affections of the spinal cord; syphilitic Pott's disease, intravertebral gummata, myelitis in a restricted sense, malignant early syphilis of the nervous tissues of the cord, acute and chronic and irregular general myelitis. He also discusses the question of hereditary syphilis, both as manifested in infancy and when delayed. The brochure is to be commended as a short and graphic description for the general practitioner conversant with the French.

JELLIFFE.

PSYCHOLOGIE DE L'INSTINCT SEXUEL. p. Jounny Roux, médecin adjoint (désigné) des asiles d'aliénés de Lyon. J. B. Baillière et Fils, Paris, 1898, 1 fr. 50.

In this, one of a new series of brochures, Dr. Roux gives an explanation of the sexual instinct which is purely mechanical. Starting on purely materialistic foundations, he shows that the function in all of its manifestations has a peripheral causative factor. The different chapters are short and discuss severally: The organic base of the sexual desire; physical love, choice and a theory of the evolution of love; superior forms of love; evolution of love, etc. This small work is quite entertaining and well worth the reading, though it contains little that is strikingly new or profound.

JELLIFFE.

DIE GESCHWUELSTE DES NERVENSYSTEMS, HIRNGESCHWUELSTE, RUECKENMARKSGESCHWUELSTE, GESCHWUELSTE DER PERIPHEREN NERVEN. Eine Klinische Studie von Dr. Ludwig Bruns, Nervenarzt in Hannover. S. Karger, Berlin, 1897.

Dr. Bruns has very modestly called his splendid monograph a "clinical study." In Eulenberg's Real Encyclopädie, Bruns contributed the article which by further study and growth has evolved to the present volume of nearly four hundred pages. It is a complete and exhaustive treatise, which bears the stamp of an authority who has made thorough researches in this field, and is a storehouse of information drawn from a rich experience.

The subject matter is divided into three portions, the first dealing with tumors of the brain, the second with tumors of the spinal cord and the third with tumors of the peripheral nerves. Each section is a monograph by itself, with full citations of the more important literature. In the first portion, under the pathology of brain tumors, Bruns distinguishes three main types. The neoplasms proper: (1) Glioma,

Sarcoma, Osteoma, Poammoma, Carcinoma, Choistestatoma, etc.; (2) the Granulomata and (3) tumors of parasitic origin. He lays some stress upon the differential diagnosis of glioma and sarcoma, the former arising, he says, from neuroglia tissue, the latter being of ectodermal origin. The second chapter deals with the occurrence and etiology of tumors, and here as an etiological factor the influence of trauma is carefully considered, the author inclining to a negative view with reference to this factor. The following chapters on the general action of tumors and the symptomatology, both general and local, are especially full and valuable. Here optic atrophy is reckoned as the most constant and important of the general symptoms of brain tumor. Of more than general value is the portion of this chapter bearing upon the localization of brain tumors, which in the author's experience was possible in at least 80 per cent. of all his cases. In the descriptions of cerebellar tumors with cerebellar ataxia the author distinguishes two types, those presenting Romberg's sign and those which do not. In chapter seven on the surgical treatment of brain tumors Bruns would seem to show anything but an optimistic feeling, in that his own experience has given few, if any, satisfactory results, yet he shows a broadmindedness in his hearty support of operative interference in tumors which can be well localized and diagnosed.

The second portion of the book on tumors of the spinal cord is as accurate and careful as the first. Here operative treatment shows far better results.

The third portion, on tumors of the nerves and the nerve plexuses, is treated of in some thirty pages. The pathological anatomy, occurrence, symptoms, prognosis, diagnosis and therapy are clearly and fully set forth.

The work is sparingly though well illustrated, and as a work for study and reference has no equal. JELLIFFE.

LES HYDROCEPHALIES. Par le Docteur Léon d'Astros. Médecin des hôpitaux de Marseille. Paris, G. Steinheil, Editeur, 1898.

D'Astros has written a voluminous treatise on hydrocephalus which is useful as giving a survey of the whole field rather than as contributing anything new to our knowledge of this somewhat obscure subject. In fact, after a rather careful search through the book, we must confess to a feeling of disappointment that we have not learned more that is new about the moot points of hydrocephalus. The book has simply taken us over the ground and presented us with a résumé of facts and opinions. In this sense it is a useful work, both for study and for reference, but it is not a work of much originality.

The author has evidently been a careful student and investigator of cerebral pathology. He is a painstaking examiner, and leaves no detail unexplored. Thus he gives not only anatomical findings, but also the chemical constituents of the cerebro-spinal fluid as a means of differentiation between the several forms of hydrocephalus. By lumbar puncture he not only secures this fluid, but he also estimates the degree of intraventricular pressure—an exact procedure which we imagine is not very generally resorted to by clinicians.

D'Astros practically limits the term hydrocephalus to the chronic internal variety of the older authors, i. e., an effusion of fluid in the ventricles, distending them and, as a secondary consequence, distending the cranium. This effusion, of course, spreads to the subarachnoid space, unless some of the natural foramina of the ventricles are pathologically closed. An *external* hydrocephalus in the older sense, i. e., an effusion limited to the subarachnoid space, but not com-

municating with the ventricles, must be, in the author's opinion, extremely rare, for reasons that are easily apparent to every one. Hence he views with disfavor an attempt to describe an external hydrocephalus in the older sense. This term, if admitted at all, should be used for those very rare cases in which the effusion is subdural, and does not communicate at all with the ventricular-arachnoid sac. In such cases, of course, the condition is radically distinct from hydrocephalus as ordinarily understood.

Beginning thus with a clear definition of his subject, d'Astros takes up seriatim all the various morbid states that cause or are associated with effusion into the ventricles.

Congenital hydrocephalus is admittedly the most characteristic and at the same time the most obscure form of the disease. The author distinguishes a true teratological and a pathological variety, but the distinction is not satisfactory, for even in the teratological form there must be practically something pathological, since this form arises from errors in the development of the embryo that are essentially morbid. This is shown by the fact that this variety is often associated with spina bifida and encephalocele. In fact, the hydrocephalus is probably due, just as is spina bifida, to a faulty development of the walls of the primitive neural tube in the embryo. It is here especially that we need new light on the radical causation of hydrocephalus, and the author, in common with others, fails to give such light. This is a chapter in embryonal pathology that is still uncompleted.

Among the various causes of acquired hydrocephalus (post-natal) d'Astros recognizes certain infections, rachitis, serous meningitis, gross lesions, such as tumors, tuberculosis, and hereditary syphilis. These causes act to produce ventricular effusion in one of two ways, either by stasis or by irritation. Stasis may be venous or lymphatic. Irritation acts especially in the vessels of the choroid plexus or in the ependyma. Effusion from a mere blocking up of the foramina of the ventricles (the foramen of Monro, the aqueduct of Sylvius, or the foramen of Magendie) is not accepted without great reserve by the author, for, as he says truly, any lesion acting to obstruct these natural orifices would probably act also to obstruct the venous outflow. This obstruction of the veins and lymphatics is a much more probable cause of effusion than a mere obstruction of the various foramina of the ventricles, through which the circulation of the cerebrospinal fluid is probably not very active.

Quinke's view of a serous meningitis is criticized by d'Astros, who does not altogether deny the existence of this disease, but very properly points out that in some recorded cases, including those of Quinke himself, the lesion is susceptible of other explanations, as, for instance, the action of tubercles or some other infection.

D'Astros concludes his book with a chapter on treatment, in which he reviews especially the various surgical procedures that have been advised for the relief of hydrocephalus. This is an especially important chapter for those who are interested in the surgical aspects of the subject, as it presents a complete view of this somewhat forlorn field of practice.

JAMES HENDRIE LLOYD.

FESTSCHRIFT ANLAESSLICH DES FÜNFZIGJAHRIGEN BESTEHENS DER PROVINZIAL-IRREN-ANSTALT ZU NIETLEBEN BEI HALLÉ. A. S. Von früheren und jetzigen Aertzten der Anstalt. F. C. W. Vogel, Leipzig. 1897.

This volume contains a number of contributions to psychiatry, both interesting and able, by alienists of international reputation.

Ueber den Querulanten-Wahnsinn, by E. Hitzig; Clinical Contributions to Forensic Psychiatry, by E. Siemerling; Reflex Epilepsy, by A. Seeligmüller; Psychical Disturbances following Attempted Suicide by Hanging, by R. Wollenberg; Uræmia Presenting the Pictures of General Paresis, by L. Bruns; Herpes Zoster, by G. Peters; Epilepsy as a Symptom of Withdrawal of Morphine, by C. Heimann; Chronic Paranoia in Epileptic Individuals, by A. Bucholz. These, with a few others, make up the contents of this excellent report. In the United States reports of this type are, unfortunately, the great exception to the rule, those usually issued from our institutions consisting for the main part of useless pages of figures. JELLIFFE.

BOOKS RECEIVED.

"The Care of the Baby," by J. P. Crozer Griffith, M. D. W. B. Saunders, Philadelphia.

"A Text-Book of Pathology," by Alfred Stengel, M. D. W. B. Saunders, Philadelphia.

"The American Pocket Medical Dictionary," by W. A. Newman Dorland, A. M., M. D.

"Cleft Palate; Treatment of Simple Fractures by Operation; Diseases of Joints, Antrectomy, Hernia," etc., by W. Arbuthnot Lane, M. D. The Medical Publishing Company, Limited, London.

"Essentials of Materia Medica, Therapeutics and Prescription-Writing," by Henry Morris, M. D. W. B. Saunders, Philadelphia.

"Ueber das Pathologische bei Goethe," by P. J. Möbius. J. A. Barth, Leipzig, 1898.

"Annuarie des Eaux Minerales Stations Climatique et Sanatoria," Paris. Gaz. des Eaux.

"Klinische und pathologische Beiträge zur Lehre von der beiderseitigen cerebralen Lähmung im Kindesalter," by W. Muratow. From Deutschen Zeitschrift f. Nervenheilkunde, 1897.

"De la Constitution des Noyaux moteur médullaires," by F. Sano.

"Abolition du reflexe rotulien malgré l'intégrité relatif de la moelle lombo-sacrée," by F. Sano. From Journal de Neurologie, 1898.

"Les Etats Neurasthéniques," by Dr. Gilles de La Tourette.

BINDING CECT. JUN 22 1966

RC

321

J78

v.25

Biological
& Medical
Serials

The Journal of nervous and
mental disease

PLEASE DO NOT REMOVE
CARDS OR SLIPS FROM THIS POCKET

UNIVERSITY OF TORONTO LIBRARY
